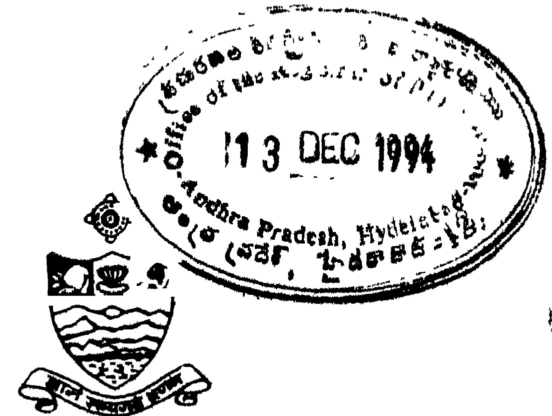
DIET AND OTHER RISK FACTORS IN ISCHEMIC HEART DISEASE

— AN ASSESSMENT

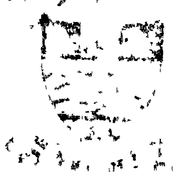
Dr. SUJATHA RAMAMURTI



SRI VENKATESWARA UNIVERSITY
TIRUPATI

1990

Published with financial assistance from the University Grants Commission



Printed at : L.V. Press, Tirupati

CONTENTS

ACKNOWLEDGEMENTS

I wish to sincerely thank and acknowledge the help of the following:

Prof (Miss) Philomena R. Reddy for her guidance, co-operation, and encouragement in the execution of this research work,

The UGC and the S.V. University authorities for their financial assistance to publish this research work,

Prof. P.V. Ramamurti, my husband for his constant encouragement and thoughtful suggestions,

Dr. D. Gunasekar and Dr. (Mrs) D. Jamuna for their unstinted co-operation and sincere assistance at various stages in the execution of this research and in the preparation of this manuscript,

Mr. L. Kullai Reddy for his help in contacting the subjects and fixing appointments,

Dr. Y. Raghava Reddy, Senior Medical Officer, S.V.U. Health Centre for help in recording the blood pressure of the subjects,

Mr. Ramachandra Reddy, Technician, S. V. U. Health Centre for help in collection of blood samples,

The non-teaching and laboratory staff of the Department of Home Science for their assistance,

Mr. Mohan who took the trouble to type the manuscript,

And, last but not the least, the 360 men who enthusiastically co-operated as subjects of the study.

SUJATHA RAMAMURTI.

INTRODUCTION

The heart is the heart of much that is life. A healthy life needs a healthy heart. A healthy heart ensures a buoyant brain! On the other hand, a diseased heart is distress to the doctor and the patient alike. And so "keep the heart healthy" became the motto of most medical men. Keep the heart healthy and you heave a sigh hearty. But, how to keep the heart healthy has been a million dollar question.

Diseases of the heart rank first among causes of death in the United states and countries of Western Europe. Most important numerically and socially are deaths due to the process of coronary atherosclerosis. Atherosclerosis, a basic pathologic process in Coronary Heart Diesase (CHD) remains for the most part an enigma in modern medicine. And rightly the World Health Organisation States that atherosclerosis resulting in Ischemic Heart Disease is the greatest epidemic that mankind has ever faced. It is a major factor in the mortality and morbidity in the developed and the developing world.

Ischemic Heart Disease (IHD) is prevalent all over the world. The International Society and Federation of Cardiology and the World Health Organization appointed a committee for standardization of the clinical nomenclature of IHD. As per their report, IHD is synonymous with the term Coronary Heart Disease (CHD). The term Ischemic Heart Disease has been defined as 'myocardial impairment due to an imbalance between coronary blood flow and myocardial requirements caused by changes in the coronary circulation. IHD comprises acute and temporary as well as chronic conditions and may be due to functional changes or organic disease. Ischemia due to non-coronary heamodynamic changes is excluded (Sainani and Mehta 1984.)

Cardio Vascular Disease is a health problem of major proportions of the population (Williams, 1969). CHD was responsible for close to 750,000 deaths per year in U.S.A. Twice as many people died from CHD as from all cancers (the second major cause of death). Its prevalence is so pervasive that clinical manifestations develop in about 20 per cent of men under the age of 60. A quarter of them die within 3 hours of first symptoms. Many never reach a hospital. Another quarter die in the first few weeks after a heart attack (Rifkind *et al*, 1979). The American heart association estimates that more than 42 million Americans (about 20% of the population) have one or more forms of blood vessel disease; more than 1.8 million persons are affected by stroke and 16 per cent of the population (an estimated 37 million persons) suffer from hypertension. About 14 per cent of adult men and 24 per cent of adult women are obese (120 per cent or more above the ideal body weight). These factors increase the risk of heart disease. Of the 1.5 million persons who are most likely to experience a heart attack in 1986, about one third may die. The estimated economic costs of heart disease and other blood vessel disorders in 1984 alone was projected as s 64.4 billion in direct and indirect costs (Posner *et al*, 1986).

Unquestionably, atherosclerosis sets the stage for IHD, but the conditions that bring about occlusion are not as well understood. Atherosclerosis appears to be a disorder of multiple etiology. Besides the local factors in the arterial wall, elevated levels of plasma lipoproteins, cholesterol and triglyceride in all probabilty appear to be important factors in the causation of atherosclerosis. Atherosclerosis is a variable combination of changes of the intima of arteries consisting of accumulation of lipids, complex carbohydrates, blood and blood products, fibrous tissue and calcium associated with medial changes. It begins in childhood, proceeds rapidly in some arteries even during adolescence and in others during early adulthood. It undergoes a series of complex changes in subsequent decades and begins to result as a clinically manifest disease during middle age (McGill *et al*, 1963).

Atherosclerosis is the main underlying cause of coronary heart disease, stroke, gangrene of extremities etc. It narrows the lumen of arteries thus producing Ischemia. Atherosclerosis leads to sudden and often complete occlusion with more severe Ischemia and resultant death of tissue in an organ such as the heart, and thus contributes to myocardial infarction (heart attack), or stroke, if it is in the brain.

The stages of development of atherosclerosis start with a fatty streak in the 2nd decade of life, progress to a fibrous plaque in the 3rd decade and produce a complicated lesion and clinically manifest disease in the 4th and 5th decades of life. Also, atherosclerosis, at least in part, is a nutritional problem. Overwhelming evidence from man and animals suggest that serum lipids, dietary habits, atherosclerosis and coronary heart disease are interrelated. The seeds of the problem are sown in the early years. Preventing fatty streaks or arresting the progression of fatty streaks is the key to this problem as fatty streaking is clinically harmless and potentially reversible (Strong *et al*, 1973).

Significant results made available from the Framingham study (Kannel *et al*, 1971) demonstrated that serum lipids and cholesterol in particular, are a predominant feature of the coronary profile and can be used to predict coronary heart disease and estimate risk. Persons with high cholesterol levels in epidemiologic studies of populations have been observed to develop coronary heart disease with much greater frequency than those with lower values, the risk being proportional to the degree of elevation of blood cholesterol (Kannel, 1974). Fidanza (1972) described that the correlation between serum cholesterol concentration and the age standardized incidence rate of all coronary heart disease cases was significant ($r = 0.84$). In every major epidemiological study preformed to date, an initial elevation of blood cholesterol was a significant predictor for the occurence of a coronary event (DeBakey *et al*, 1986).

Triglycerides (TG) are the major lipids in the diets of human beings and represent an important source of fuel in the living body. These are esters of fatty acids and glycerol and are

commonly referred to as neutral fat. TG is one of the two blood lipids with which the dietitian, the physician and the patient are most frequently concerned, the other being cholesterol (Gotto and Scott, 1973). The stockholm study's findings suggest that triglycerides carry a risk independent of cholesterol. Increase in triglyceride and cholesterol combined, constituted a greater risk than either one alone (Albrink, 1973). A mean triglyceride of 65 mg/dl in subjects below the age of 20 years rises progressively to about 95 mg/dl in the 6th decade. Values above the 160–200 mg/dl range are considered abnormal (Tzagournis, 1978).

Serum triglycerides are an independent additional factor for the development of coronary arterial disease. In a nine year follow-up study of 3168 men, the rate of coronary disease increased linearly with increasing triglycerides or cholesterol levels and the combined elevation of both lipids carry the highest risk. The increase in rate of coronary disease was 4.9/1000 for each quintile of increasing triglyceride level and 4.1 per 1000 for each cholesterol quintile (Tzagournis, 1978). Hypertriglyceredemia may also contribute to Atherosclerotic Cardio Vascular Disease (ASCVD) by mechanisms independent of atheroma formation, by causing symptomatic expression of ASCVD as Angina Pectoris or as sudden death. Several lines of evidence support hypertriglyceredemia as an agent involved in such symptomatic expression, the mechanism of action being unclear (Albrink, 1973).

Investigative and exploratory researches on coronary Artery Disease revealed a number of risk ractors. Of these, the ones that have direct nutritional implications are hyperlipidemia, hypertension and obesity. The other important risk factors that have been documented by many others are cigarette smoking, heredity and physical inactivity. All these risk factors are associated with susceptibility to Ischemic or coronary heart disease. The risk factors and susceptibility to heart disease vary from race to race and from one community to another as they are influenced, not only by hereditary or constitutional factors, but also by environmental and cultural factors.

Diet is an integral part of culture. Food habits vary tremendously from country to country and even in the same country, from one state to another. The nutritional factors play a significant role in the causation of atherosclerosis, affecting the blood lipid levels to a great extent. Based on clinical, epidemiological, pathological and laboratory animal studies, there is considerable evidence which relates diet and the levels of certain blood lipids to the development of atherosclerosis and its clinical complication, notably Ischemic heart disease (Sirtori *et al*, 1975). Comparison of population groups have shown a strongly positive correlation among the intake of dietary cholesterol, saturated fat and total plasma cholesterol and atherosclerotic disease mortality (Mcgill, 1979). A positive relationship has been indicated between intake of total fat expressed as per cent of total calories with atherosclerotic lession involvement in the human being (Devadas and Eswaran, 1979, Moore *et al*, 1977).

Ischemic heart disease is as common in India as in the developed countries. The incidence of heart disease in India, as reported by Pinto *et al* (1970) varied from 11 to 23 per cent. According to the report of Sarvotham, the prevalence rate among males of Chandigarh (North India) was 65.4 per thousand which was nearly the same as in Tecumseh, U.S.A. A study of people over 30 years of age conducted in a sector of the city of Bomba composed of semi-skilled workers, showed a prevalence of 2 cases per thousand (Pinto *et al*, 1970). A review of the statistics of the Government General Hospital, Madras (South India) showed that Myocardial infarction (heart attack) is the number one killer (about 40%) among the medical emergencies (Senthilanathan, 1979).

IHD affects the people at the prime of their life and at the peak of their career when their services are most needed for the country, and their families. A developing country like India cannot afford to lose the intelligentia at a time when they are at the crest of their careers. Unfortunately, Myocardial Infarction often occurs without any warning. The first attack itself may be a fatal one, in about 40 per cent of cases (Senthilanathan, 1979).

According to Sapru (1984), the total number of patients of Coronary Artery Disease (or IHD) in India at present would be at least 3.45 million. Even the lowest estimate of 1.04 per cent (age group 25 years and above) would suggest that there are 2.78 million cases of IHD in the country. Sapru (1984) believes that most of his professional colleagues would consider this figure to be a serious underestimate. Considering the decadal growth in population, the number of new cases of IHD appearing each year during the present decade would be 190,000.

Almost all individuals including the poor socioeconomic groups have atherosclerosis of the coronary arteries, which is the forerunner of manifest Coronary Artery Disease after the age of 40 years. During 1979, 9.7 per cent of all deaths in the rural population were due to cardio-vascular disease (CVD) and this is likely to be higher in the urban population. Of the entire population in the country, 21.89 million or 3.3 per cent are presently suffering from CVD. When the mortality data is compared against that of U.S.A., Australia and Japan, India also emerges high on the list. This poses a formidable problem and needs to be tackled without further loss of time (Sapru, 1984).

The food habits and eating patterns which form an integral part of culture and tradition vary from one state to another and from one region to another (National Nutrition Monitoring Bureau, (NNMB, 1981). The variegated cultures of India and other factors like economic status, geographic location, race, religion and rearing practices influence the ultimate eating behaviour of people. Yet, amidst the cultural diversity that is India, there are common threads that bind the people together in their food practices. Indian diets are mainly cereal based. But the recipes can vary. Wheat, rice and millets are the staples which supply considerable amount of carbohydrates. There is difference in the quantity and quality of various dietary components in the Indian diets as compared against the Western diets. It would therefore be useful to study the relationship between various dietary constituents and the lipid fractions like cholesterol and triglycerides in the Indian population.

It is well documented in the literature that there exists a significant association between the elevated levels of blood lipids, particularly cholesterol and triglyceride and the incidence of IHD A major chemical constituent of the atherosclerotic coronary arteries in both man and animals is cholesterol (McGill, 1974). Elevated levels of cholestrol and/or triglycerides in blood are believed to result in more rapid and severe development of atherosclerosis and in turn to Coronary Heart Disease (Devadas and Easwaran, 1979).

There is widespread agreement that atherosclerosis is a multifactorial disease i. e. more than one factor appears to be implicated. Several risk factors appear to be associated, of which the major ones include serum cholesterol, high blood pressure, cigarette smoking and obesity. Certain personality traits, family history, lack of exercise, age and gender (being male) are some of the other risk factors (Bordia and Arora, 1974, Chandra Patel, 1983, Whitney and Cataldo, 1983) Each of these risk factors has been associated with increased lipid levels in different clinical investigations. Each contributes to susceptibility to heart disease. Most of the studies on these relationships were conducted in the west. There is a dearth of similar studies carried out in India. Even the few studies that have been reported were mostly on hospital populations. A few, carried out on non-hospital populations included only limited variables. (Singh *et al*, 1980; Devadas *et al*, 1980; Gandhi, 1982). Also, studies relating dietary constituents to serum cholesterol and especially to serum triglyceride levels have been few, if any. As nutritionists, the concern lies mostly in the evaluation of these relationships.

It is in the context of the foregoing, that the present study was planned to assess cholesterol and triglyceride levels in subjects of three age groups i.e., 20–29, 30–39, and 40–55 years. The relationship of other coronary risk factors like dietary constituents, age, obesity, hypertension, cigarette smoking, family history and coronary prone behaviour to serum cholesterol (SC) and serum triglycersde (STG) levels was studied independently and in combination. More specifically stated, the objectives of the present study were as follows:

1. To estimate SC and STG levels in a sample of student, teaching and non-teaching population of Sri Venkateswara University, belonging to 20–29, 30–39 and 40–55 years age groups.

2. To assess the mean nutrient composition of the food intake of these subjects.

3. To examine the relationships of each one of the dietary constituents to SC and STG levels.

4. To assess the association of each of the risk factors namely age, obesity, hypertension, cigarette smoking, family history and coronary prone behaviour to SC and STG levels.

5. To study the combined contribution of the aforesaid variables to SC and STG levels.

A REVIEW OF SOME RELATED STUDIES

Since the beginning of the century, attempts to understand the causes of Ischemic Heart Disease (IHD) have been growing in geometric proportions. More men, more money and more methodological sophistication has been a salient feature of this development. In the result, there is a sea of articles and scores of treatises comprising almost every conceivable factor that may seem to associate with IHD. To review even a moiety of these would not only be an impossible task but also would constitute an unwarranted ambition.

In as much as this investigation limits to an examination of a few of the more important factors in relation to the two blood lipids, serum cholesterol and serum triglycerides in an Indian context, the review would be accordingly restricted. Further, being fully aware of the constraints of working in a developing country with a belated availability of more recent researches, no claim is made of comprehensiveness or completeness. Presented are representative findings of relevance, relating to the factors under investigation.

The material is reviewed in three sections viz.,

1.1 IHD and the blood lipids-serum cholesterol and serum triglycerides.

1.2 Risk factors other than diet in IHD.

1.3 Diet in relation to blood lipids.

1.1 Ischemic Heart Disease and Blood Lipids.

Ischemic Heart Disease (IHD) was a rarity before 1900. The increase in its incidence since then resulted in considering it as a major epidemic. Giving a critical analysis of the statistical data

on IHD, Slater *et al*, (1985) stated that a true epidemic of acute IHD disease has occurred, affecting males either exclusively, or to a greater degree than females and is now on the decline. But, IHD is an epidemic whose exact magnitude may never be resolved.

IHD is defined as a cardiac disability, acute or chronic, arising from reduction or arrest of blood supply to the myocardium in association with disease processes in the coronary arterial system. IHD is synonymous with the term Coronary Heart Disease (CHD). As per the clinical nomenclature, IHD has been defined as myocardial impairment due to an imbalance between coronary blood flow and myocardial requirement caused by changes in the coronary circulation, IHD comprises of acute, temporary, as well as chronic conditions (Sainani and Mehta, 1984).

1.1.1 Prevalence of IHD in India

Some years back it was believed that IHD was comparatively rare in developing countries like India. But now this opinion is being revised as a result of the findings in various cardiological centres in India. It is found to be common in the developing countries also (Senthilnathan, 1979).

Few epidemiological studies of IHD have been carried out in India using strict clinical and electrocardiographic criteria for diagnosis. According to Pinto *et al* (1970) the incidence of IHD in different parts of India varied from 11 per cent to 23 per cent between 1941 and 1966. Pinto *et al* (1970) quoting Sarvotham who surveyed 2030 persons in Chandigarh (North India) stated that the prevalence rate of IHD of 65.4 per thousand was nearly the same as in Tecumseh, USA.

A study of pepole over 30 years of age conducted in a sector of the city of Bombay, comprising semiskilled workers, showed a prevalence of 29 cases per thousand. The total sample of 569 constituted 25 per cent of the people residing in that area. The prevalence of IHD in the age group below 40 was 24 per

thousand in the Chandigarh series. This illustrates the fact that the proportion of young patients with IHD is fairly high in India (Pinto *et al*, 1970).

Dhar *et al* (1978) presented information with regard to the pattern of heart disease. The per cent incidence of IHD was 4.4 and 7.4 in the 1965–1967 and 1973–1975 periods respectively. Between the sexes, the incidence was more in males.

Sapru (1984) giving the incidence of Cardio Vascular Disease, states that IHD and hypertension is prevalent in India to a considerable extent. 21.89 million people in the country (India) at present are victims of cardio vascular diseases (CVD) and that to this pool approximately 1.12 million are likely to be added each year. During 1979, in the rural population, approximately 9.7 per cent of all deaths were due to CVDs. Mortality from these diseases in the urban population is likely to be higher.

1.1.2 Pathology of Atherosclerosis:

The basic pathological problem in IHD or CHD is atherosclerosis. A general term used to describe thickening and hardening of the arteries is Arteriosclerosis. When this process is due to the accumulation of lipid plaques within the arterial wall, the condition is known as Atherosclerosis.

Atherosclerosis is a disease which affects the inner most portion of the lining of arteries (Tunica intima) and which ultimately invades the middle coat (Tunica media). The lesion known as an atheroma, consists of a collection of intracellular and extracellular lipid, accompanied by proliferation of cells, by accumulation of mucopolysaccharides and by changes in the fibrous components of the arterial wall. The accumulated lipid is dominated by the esterified cholesterol (Seymour Dayton, 1975). Two theories have been put forward to explain the pathogenesis of atheroma. The first emphasises infiltration of plasma lipids, presumably in the form of lipoproteins and the second lays emphasis on fibrin deposition and fibrinolysis. Both processes may be involved i.e. the lesion is initiated as a fibrin thrombus, but the lipid enters from plasma, across the

endothelium. The initial lesion is thought to be small, an exclusively intimal accumulation known as a fatty streak, consisting primarily of lipid–laden microphages, known as foam cells, within the intima. This is thought to progress by stages to a fibrous plaque i.e. a lesion still lipid rich, in which a great deal of connective tissue has proliferated (Seymour Dayton, 1975).

The term arteriosclerosis signifies a palpable and visible pathological condition of the vessels, namely the hardening and thickening of the wall. It is a disease of the intima depending on the chemistry of the blood on the one hand but on the other, also on the structure and metabolism of those layers of the vascular wall which are supplied by the blood. It leads to a thickening of the intima. With the absorption of substances (water, protein, lipid, mucopolysaccharides and minerals) and accumulation of deposits, the innercoat stretches, as a result of which, the lumen, to a large extent narrows considerably. This is the central clinical problem, because individual organs become diseased due to a local circulatory insufficiency thus brought about. It is fundamentally a localised lesion of the wall. It is a disease punctuated by time. It's progress is imperceptible in the initial stages and it is intermittently progressive (Schettler and Boyd, 1969).

Fatty Dots and Streaks : The first of these appear as a yellow (fatty) dot or streak. In the early yellow dot, fat droplets are present in the native smooth muscle cells of a focal area. As the lesion grows in size, the number of fat droplets in a given smooth muscle cell, as well as the number of these cells involved in the change, increases. When much of the cytoplasm is occupied by fat, the cell acquires the appearance of a foam cell. Cellular necrosis progresses with advancement of the lesion. The earliest lesion seen as an intimal lipid deposition (the fatty streak) is converted into an elevated lesion with a lipid core and a fibromuscular cap (the fibrous plaque).

Chemical analysis of the lipids show that the cholesterol ester is the dominant lipid, 81 per cent of total lipids in fatty streaks and 59 per cent of total lipid in fibrous plaques. The

proportions of ester and other lipids are distinctly different for the fatty streak and fibrous plaque inclusions (Schettler and Boyd 1969).

The fibrous plaque undergoes a variety of changes, some of which result in arterial occlusion. Ischemia of vital organs results in various clinical disease syndromes depending on the organ affected. When one or more of the risk factors for clinical diseases are present, some juvenile fatty streaks undergo changes that eventually result in the formation of fibrous plaques. Prevention should be aimed at the advanced fatty streak and the fibrous plaque, which begin to develop at about the beginning of the third decade of life (McGill, Jr. 1974).

Risk factors which promote CVD are a constellation of influences. Some, such as those determining susceptibility (e.g. age, sex and heredity) we can do little about. Atherogenic personal attributes such as, serum lipids and blood pressure are modifiable. Living habits that promote these traits such as, faulty diet, overeating and physical inactivity are also modifiable Other habits like cigarette smoking which can precipitate coronary attacks are also theoretically correctable. These factors have been shown to be powerful contributors to risk under age 65, the risk mounting in proportion to the number and the level of these contributing factors.

1.1.3 Hyperlipidaemia

Of the multiple risk factors associated with Ischemic Heart Disease, Hyperlipidaemia ranks first. A convincing relationship between blood lipid concentration especially cholesterol and atherogenesis has been shown. This is based on the following facts. a) Severe atherosclerosis is the underlying pathogenic process in most clinical cases of IHD. b) A several fold increase in cholesterol particularly esterified cholesterol is the biochemical hallmark of the atherosclerotic plaque. c) The excess cholesterol in the plaque is derived from the cholesterol-bearing lipoproteins of the circulating plasma. d) Sustained hypercholesterolemia or hyperlipidaemia is associated with

severe atherosclerotic CHD. e) In groups of middle-aged patients with CHD, higher mean serum-cholesterol-lipid-Betalipoprotein levels are found than in matched control groups. f) Where the mean serum cholesterol levels of populations are low, clinical CHD and severe coronary atherosclerosis at post-mortem are rare, particularly in middle age (Raab, 1966).

On the basis of the data from the now classic Framingham study, Kannel (1977[b]) brought out in clear terms the relationship between the serum cholesterol level and the incidence of CHD (Table-1) with increase in serum cholesterol level there was a substantial increase in the 22 year incidence rate of CHD.

Table 1 : Twenty Two Year Incidence of Coronary Heart Disease According to Serum Cholesterol Level–Framingham Study, Men Aged 30 to 39.

Serum Cholesterol Level	Population at Risk	CHD Cases	22 Year Incidence Rate per 1,000.
Less then 200 mg/dl	319	22	72.5
200 – 219	128	12	97-6
220 – 239	169	29	183.4
240 – 259	97	19	217.7
More than 259 mg/dl	119	35	335.4

Hyperlipidaemia is defined as excess concentrations of cholesterol or triglyceride, or both in plasma. Most practitioners would accept, as raised cholesterol concentration, values

above 6.5 m mol/1 (251 mg/dl) and as raised triglyceride concentrations values above 1.5 m mol/1 (133 mg/dl) for females and 2.1 m mol/1 (186 mg/dl) for males (Murchison, 1985). In practice, the decision of how far to investigate and treat hyperlipidaemia with the aim of preventing Ischemic Heart Disease is based not only on serum lipid concentrations, but also on considerations such as the age of the patient, family history of early onset of atherosclerotic disease and the presence of other risk factors such as hypertension.

All of the plasma lipids–cholesterol, triglyceride and phospholipids are solubilized and transported as macromolecular complexes referred to as plasma lipoproteins. There are 4 major families of the plasma lipoproteins. The largest of these particles, the chylomicrons, transport practically all of the dieetary or exogenous triglyceride. The next largest family is the very Low Density Lipoproteins (VLDL), also called the pre-Beta lipoproteins because of their mobility in front of the Beta Globulins on electrophoresis. The VLDL transport the endogenous triglycerides, which are synthesized mainly by the liver but to some extent by the gastro-intestinal tract. The third family lipoproteins are the Low Density Lipoproteins (LDL) or beta lipoproteins. This family transports half to 2/3rd of the total plasma cholesterol Finally, the High Density Lipoproteins (HDL), or alpha–lipoproteins are the smallest of the families and contain approximately equal amounts by weight of protein and lipids.

Since the lipoproteins represent the functional transport carriers of all plasma lipids, it is more specific and meaningful to translate hyper–lipidaemia into the term hyper–lipoproteinaemia. For purposes of screening a general population for overall assessment of risk of atherosclerosis, a simple determination of cholesterol, or triglyceride is just as adequate as the measurement of the concentrations of the individual lipoproteins (Gotto and Scott, 1973).

1.1.3.1 Cholesterol

Cholesterol, a steroid alcohol with the formula $C_{27}H_{45}OH$ is the precursor of bile acids, steroid hormones and pro-vitamni D_3. Substantial amounts of total body cholesterol are obtained

from the diet even though a major portion of the cholesterol needed for normal body functions is endogenously synthesized. It is present in the blood where it plays an important role in the transport of fatty acids. It is present in all foods of animal origin. The discovery of cholesterol is attributed to Nichel Eugene Chevreul of France, who, in 1812 first differentiated between saponifiable and non-saponifiable lipids. The correct structure of cholesterol was finally established in 1932 (McGill, 1979).

The greater part of the cholesterol of the body arises by synthesis (about 1 gram/day), whereas only about 0.3 g/day are provided by the average diet. Cholesterol is eliminated via two main pathways: conversion to bile acids and excretion of neutral sterols in the feces. Cholesterol is readily absorbed from the small intestine, where, most of cholesterol ester is hydrolyzed to free cholesterol and fatty acid as a result of the action of pancreatic cholesterol esterase. The peak cholesterol absorption takes place in approximately 6–9 hours.

The capacity of cholesterol absorption mechanism is limited, and dietary cholesterol must compensate the cholesterol excreted via the bile ducts. Dietary fat affects cholesterol absorption by stimulating the flow of bile and by providing fatty acids for cholesterol esterification. Fat may improve micellar solubilization. Cholesterol esters play a major role in cholesterol absorption and bile salts play an even more important role in stimulating the absorption of cholesterol than the solvent action of fat (Whol and Goodhart, 1968).

Liver is a major site of cholesterol biosynthesis and degradation. Cholesterol is excreted by the liver in bile. The liver therefore, has a major cholesterol regulating role. Cholesterol which is needed to form cell membranes, bile salts and steroid hormones is derived from the diet and endogenously synthesized from the acetyl CoA pool.

Cholesterol synthesis could be controlled and in this the liver plays a major role. In humans, extrahepatic synthesis, mainly in the intestine, is more important. Bile acids inhibit

cholesterol synthesis in the intestine. Attempts to lower plasma cholesterol in humans by reducing the amount of cholesterol in the diet are more effective. An increase of 100 mg in dietary cholesterol causes a rise of 5 mg cholesterol per 100 ml serum (Martin *et al*, 1981). In humans, the total plasma cholesterol is about 200 mg/dl, rising with age, although there are wide variations between individuals.

Many investigators have demonstrated a correlation between raised serum lipid leved and the incidence of CHD and atherosclerosis in humans. Of the serum lipids, cholesterol has been the one, most often singled out as being chiefly concerned in the relationship. However, other parameters like lipoproteins, serum triacyl glycerol concentration etc. show similar correlations (Martin *et al*, 1981).

Most patients can be classified for clinical purposes, on the basis of serum cholesterol and triglyceride concentrations and a stored plasma test. An increase in serum total cholesterol usually indicates increased concentrations of LDL and Lipoprotein. Most hyper lipidaemic subjects owe their abnormality to a complex interaction of polygenic and environmental factors (Murchison, 1985).

Plasma lipids and lipoproteins are major risk factors for CHD. Cholesterol and LDL levels are strongly and directly related to CHD risk. It was found that the risk of a myocardial infarction for men between 30 and 49 years is increased more than five fold, if serum cholesterol is greater than 260 mg/100 ml as compared to a man with a value less than 220 mg/100 ml (Gotto and Scott, 1973). Based on SC levels of children, it was speculated that a level 200 mg/100 ml in a 6 year old boy may represent a risk equivalant to that predicted by a value of 238 mg/100 ml in an adult.

Increased risk to CHD starts at about 220-230 mg per dl of serum cholesterol (Reiser, 1984). The Lipid Research Clinics program presented reference values for hyperlipidaemia. The prevalence study was carried out on 60,502 Americans. The

mean reference values for plasma cholesterol ranged from 165 mg/dl in 20–24 age group to 215 mg/dl in 40–69 age group in white males (Rifkind and segal, 1983).

The 75th and 90th percentiles were also provided to indicate that any one in the top decile or even quartile of the distribution is at considerably higher risk for the development of CHD and hence, should not be regarded as having desirable lipid concentrations.

Rifkind and Segal (1983) comment that reference values may vary, sometimes notably, from population to population. However reference values continue to be used, since they identify persons at especially high risk for CHD as compared to their peers of the same sex who have lower lipid levels. These values also help in screening those with a genetic form of hyperlipidaemia. Men in the upper quintile (80th percentile) of the serum cholesterol distribution accounted for more than half of the excess coronary events attributable to hypercholesterolemia.

The risk of CHD increasing directly with plasma cholesterol (PC) level is evident even in cross-cultural studies. The diversity of mean cholesterol among different populations is largely attributable to diet. Also there is a wide range of variation of PC within single populations, but the reasons for within population variation in PC are far less apparent. A singificant but small part of the variance is due to individual differences in diet. Genetic, probably polygenic determinants provide a further partial explanation, though the mechanism of such genetic effects is unknown.

4.13.2 The Triglycerides

Triglyceride is the major lipid in the diet of human beings and represents an important source of fuel in the living body. Triglycerides are esters of fatty acid and glycerol (Vranic, 1975). A simple triglyceride is one in which the three fatty acids are the same. A mixed triglyceride is one in which at least two of the

fatty acids are different. This group accounts for about 98 per cent of fats in foods and over 90 per cent of the total fat on body (Robinson, 1977).

Endogenous Triglyceride Synthesis: Exogenous triglycerides are not the only source of triglycerides in the blood, Endogenous Triglycerides (TG) are transported as VLDL, Endogenous TGs may be derived from reassembled exogenous fatty acids, FFA from adipose cells or metabolic products of ingested glucose or protein. VLDL are a large group of macro molecules synthesized and secreted mainly by the liver. They contain large quantities of triglycerides (TGS) assembled with small amounts of cholesterol, phospholipids and apoproteins B as well as apoproteins C series. In the sequential delipidation of the VLDL, TGs and the C apo proteins are removed in a step-wise fashion. The VLDL is made up of progressively smaller molecules with diminishing TG and C apoprotein content as lipoprotein lipase acts upon them. The end product of VLDL metabolism is LDL with apoprotein B as the major protein unit. Many cells have the ability to bind LDL and utilise the cholesterol and phospholipid for menbrane synthesis. Apoprotein B is critical in regulating cholesterol synthesis by influencing a key enzyme hydroxymethyl-glutanyl COA reductase. The C apoproteins are transferred io HDL during metabolic degradation of VLDL. HDL with its major protein unit, apoprotein A, affects the activity of lecithin–cholesterol acyl transferase, another important lipid regulating enzyme. An improved understanding of overall lipid metabolism has resulted from the studies of endogenous TG disposition (Tzagournis, 1978).

The synthesis of TG consists of the successive additions of acyl moities from 2 moles of acyl COA to Lglycerol phosphate to form phosphotidic acid which is dephosphorylated by phosphotidic acid phosphatase to from 1, 2-diglyceride. The addition of another acyl group from acyl COA results in a molecule of triglyceride. (Rober Scheig, 1974).

There is an increased risk of atherosclerosis with elevated endogenous triglyceride, but it is not clear if this is due to

triglycerides per se, or the cholesterol content of the common lipoprotein that transports both lipids.

Plasma TG originate from 2 chief sources viz., large particles derived from dietary fat delivered through the gut (the chylomicrons) and the particles endogenously synthesized by the liver and secreted into the circulation as somewhat smaller particles but still large enough to cause turbidity of serum. The feeding of a fat meal of any type causes only minimal rise in TGS due to increase in circulating chylomicrons. Elevation of chylomicrons for more than 6 to 8 hours is unusual; therefore chylomicronemia after an overnight fast is rarely a cause of significant hypertriglyceredemia.

Dietary fat intake is almost never a cause of hypertriglyceredemia (Hy TGa). In the rare instances in which it is, the particles are not atherogenic. The small endogenous particles, in contrast, are responsible for most clinical HyTGa. These particles are smaller than chylomicrons and higher in cholesterol. They are present after an overnight fast when chylomicrons have disappeared. These endogenously synthesized TGs are associated with increased risk of Hy TGa (Albrink, 1973).

The most important factors influencing endogenous TG concentration are carbohydrate intake, obesity and heredity. TGs play a role in the body economy by serving as a repository for excess calories in adipose tissue. In man, when the excess calories are in the form of carbohydrate, the glucose is first converted into TGs in the liver and secreted into the circulation as VLDL, then transported to adipose tissue, where, under the action of lipoprotein lipase it is incorporated and stored as fat. A very high carbohydrate diet, in which carbohydrate contributes 75 per cent or more of calories, causes an almost universal increase in TG concentrations of uncertain duration, perhaps a permanent elevation in susceptible individuals. Increased hepatic synthesis of TG from carbohydrate is the probable cause. This type of lipemia thus represents over production of TGs. In general, TGs are approximately doubled by a higher carbohydrate diet. Sucrose causes greater HYTGa than does starch.

Two theories are put forward to explain basal HYTGa. Firstly, a high TG synthesis from carbohydrate occurs, even with normal arbohy drate intake and secondly, an impaired removal of TG, the removal defect merely being exaggerated by a high carbohydrate diet (Albrink, 1973).

Serum TGs are affected by diverse underlying disorders. Several factors play a role in the pathogenesis of HYTGa including diet, body weight, genetic influences and glucose metabolism Normal serum concentrations tend to rise with age, i.e., from 65 mg'100 ml to 95 mg/100 ml from 20 to 60 years. Values above the 160-200 mg/100 ml range are considered abnormal (Tzagournis, 1978).

Kannel (1977[b]) stated that ideal values for cholesterol are probably below 200 mg/100 ml of serum. Within the range of cholesterol values reported as normal by most clinical laboratories (180-310 mg/100 ml), there is a five fold range of cardio vascular risk. Cholesterol levels exceeding 250 mg/100 ml and triglyceride levels exceeding 200 mg/100 ml are reasonable working definitions of hyperlipidemia for preventive cardiology.

1.1.3.3 Indian Studies on Serum Cholesterol and Serum Triglyceride Levels.

Pinto *et al* (1970) compared the serum cholesterol (SC) levels as reported by Indian and Western authors. According to the Indian studies, the SC values were low in the age group 21–30 years, but increased with age. The SC values of 152–174 mg/dl, in Indian studies were similar to those of populations from different parts of India, but were much lower than normal levels reported by Western workers. According to Gertler, Keys and Kannel the SC levels were above 200 mg/dl and they increased with age in the normal population. SC values increased with age in any group and they were higher for men than women.

Serum cholesterol level is iufluenced to a very great extent by dietary constituents which in turn, depend on the economic status of the individuals. Srikantia *et al* (1961) conducted a study on some South Indian population groups varying in

income. A positive correlation between serum cholesterol and income group was shown. As the income of the group increased the serum cholesterol level of the subjects also increased. This may be attributed to the habitual use of rich foods consumed by affluent familes. Cholesterol concentrations progressively increased with advancing age among the people of the high income groups. The low income groups in Trivandrum and Nilgiris were consuming coconut oil and sesame oil respectively, the total fat intake being low in both thc groups i.e., not more than 10 to 12 per cent of the diet. However, the high income groups used hydrogenated vegetable fat. The highest levels of serum cholesterol i.e., around 229 mg per cent were observed among the officers of the Defence services.

High serum cholesterol levels among people of high socio-economic group were also reported by Devadas & Eswaran (1979) who suggested that there is a slight shift of SC values toward the higher ranges among the rich when compared to the poor. In an epidemiological survey carried out in Punjab (Werners and Sareen, 1978) the SC levels of 3057 persons belonging to the middle or lower socio-economic groups were estimated. The values varied according to age. It was 133.46 mg/dl for adolescents, 160.82 mg/dl for adults and for persons over 50 years of age it was 181.02 mg/dl. Higher levels were recorded for three smaller selected groups. Among the obese persons (N=327) the mean values were around 20 mg/dl higher than those of non-obese persons in each of the age groups. In the age group 30-50, there were 71 subjects (Males and Females) belonging to the higher classes with mean value of 204.25 ± 7.3 and in the 50 plus age group, there was 77 subjects belonging to the higher classes with a mean value of 232.85 ± 7.05 mg per cent. There were 311 patients with diseases of the cardiovascular system, who had a mean cholesterol level of 215.45 mg/dl.

The relationsnip between blood lipids and IHD has been widely discussed. Several studies have concluded that a high cholesterol is an important risk factor. In view of the marked differences in blood lipid levels around the world, due to social, economic and nutritional factors, it is necessary to define reference ranges. To define reference ranges for serum cholesterol (SC) triglycerides (STG) and other lipoprotein fractions in South India, a study was conducted on hospital patients with Ischemic Heart Disease and controls (Barrington *et al*, 1980). The reference ranges for serum lipids are given in Tables 2 and 3.

Table 2: Reference Ranges for Serum Cholesterol and Serum Triglycerides

Constituent	N	Refeerence values (mg per cent) Mean	Refeerence values (mg per cent) Range	Probit Analysis Mean	Probit Analysis Range
Cholesterol	279	185	115–255	175	100–250
Triglyceride	350	124	52–196	111	43–179

Table 3 : Variations of Cholesterol and Triglyceride with Age

Age Group (Years)	0–19	20–29	30–39	40–49	50–59	60+
Cholesterol						
Number of subjects	18	23	24	33	34	27
Mean mg/dl	164	180	187	195	194	198
Reference Range	102–226	97–263	130–244	108–282	95–293	125–271
Triglyceride						
Number of Subjects	18	22	24	31	29	25
Mean mg/dl	106	108	124	128	124	132
Reference Range	22–90	39–177	45–203	58–198	47–210	66–198

The data presented in Table 3 shows that the cholesterol and triglyceride values increased with age upto age 40–50 for TG However, statistically significant differences were not observed by the authors between the IHD and control groups, the triglyceride and Pre Beta lipoprotein values tended to be higher in the IHD group.

Devadas *et al*, (1980) estimated the serum cholesterol levels of thirty Tamilian and Gujarati women in the age range 25–40 years. The estimated SC values were 192, 225 and 226mg per cent for the Tamil vegetarians, Tamil Non-vegetarian and Gujarati vegetarian women respectively (N = 10 in each category).

Yet another endeavour was made to assess the normal value of serum cholesterol in male and female subjects (Singh *et al*, 1980). Total serum cholesterol levels of 114 young healthy subjects (80 males and 34 females in the 18–22 age group) were studied and is presented in Table 4.

In the case of males, the total SC level ranged from 143 to 300 mg per cent with an average value of 205.3 ± 28.79 mg per cent. In the case of females it ranged from 141 to 234 with an average of 186.0 ± 25.2 mg per cent. The difference between the sexes was statistically significant ($P<0.01$). It clearly shows higher level in male subjects as compared to the females. Majority of the subjects were in the range of 181–210 mg followed by 34 subjects in the range of 211 to 240 mg per cent. By percentage, 91.2 per cent of the subjects fell in the range of 151 to 240 mg per cent which is close to that observed by Varley (1975).

Table 4: Distribution of the Sample According to Their Total Serum Cholesterol Levels

Subjects	Total Serum Cholesterol Level (mg per cent)							
	150	151–180	181–210	211–240	241–270	271	Mean ± SD	t
Males (80)	2.4	17.5	40.0	32.5	5.0	2.5	205.3 + 28.7	
N	(2)	(14)	(32)	(26)	(4)	(2)		
Females (34)	5.88	35.28	35.28	23.56	–	–	186.0 ± 25.7	3.58* P<0.01
N	(2)	(12)	(12)	(18)				
Total (114)	3.5	22.8	38.6	29.8	3.5	1.8	190.5 ± 29.1	
	(4)	(26)	(44)	(34)	(4)	(2)		

(The numbers are in percentages)

In a similar investigation Gandhi (1982) studied the lipoprotein composition of healthly normal control subjects from North India in relation to age and sex and compared them with Western data. A total of 201 normal healthy subjects of both sexes and of different age groups were studied. These values for males in comparison with Western values (Fredrickson *et al*, 1967) are given in Table 5.

Table 5 : Comparison Between Indian Population (controls) and Western Population (controls males) for Differences in Lipids

Age Years	Population	Male		
		N Mean	Plasma cholesterol Values in mg/dl	Plasma Triglycerides ± SD
0–20	Indian	41	133 ± 20	119 ± 24
	Western	43	172 ± 34b	61 ± 34a
21–30	Indian	36	159 ± 28	126 ± 35
	Western	41	183 ± 37b	73 ± 32a
31–40	Indian	22	170 ∓ 21	124 ± 20
	Western	50	210 ± 33a	78 ± 39a
41–50	Indian	27	178 ± 22	139 ± 29
	Western	67	230 ± 55a	90 ∓ 41
51–60	Indian	11	187 ± 17	145 ± 22
	Western	28	240 ± 48b	104 ± 45a
Overall mean	Indian	201	157 ± 29	124 ± 29

P values: $a<0.001$; $b<0.01$; $c<0.05$

Total cholesterol increased with age in both sexes. The mean cholesterol value for males was 160 ± 29 and the mean triglyceride values were 128 ± 29 mg/dl. Values in males were higher than those of females and the overall difference was significant.

When the Indian data was compared with the western data by the author, it was observed that cholesterol was significantly higher in the western population, while triglycerides were significantly higher in the Indian population. VLDL cholesterol concentration which may be used as an indication of high triglycerides was more in the Indian population.

The differences between the populations could be due to environment and diet. Indian diets are mainly in the form of carbohydrates from pulses and cereals which could be the reason for higher triglyceride values. Western diets are rich in eggs, fats and oils and flesh foods. This might have increased their SC values. Indian foods rich in crude fibre could also be a reason for lower cholesterol values in the Indian population. Gandhi (1982) stated that in view of the fact that the number of subjects in his study was small and that the diet and seasonal variations were not taken into account, the values obtained cannot be considered an established standard of reference.

Tasker *et al* (1983) assessed the lipid profile in patients with myocardial infarction in the city of Bombay. According to their results, the Indian patients with CHD have lower levels of lipids than do the western population. 54 per cent of the patients had their lipid levels within normal limits while 45 per cent had higher lipoproteinemia. In the latter group 43 per cent showed type II b & III pattern and 22 per cent showed Ia pattern. The MI patients in this study showed statistically lower HDL–C values compared to control. The authors suggested that this may be due to high carbohydrate content of the diet which may also explain a high incidence of Type II b followed by IV and Type IIa. This distinct difference in the Indian population from the western pattern in which type IIa is predominant, is presumably due to high carbohydrate and low fat diet.

Murthy *et al*, (1986) reported the serum cholesterol and triglyceride levels of 55–64 year old men and women. The subjects were apparently normal. The serum cholesterol level of males was 229.4 ± 3.36 and of females 220.0 ± 17.65 mg/dl. The serum triglyceride level was 95 82 ± 42.04 and 102.62 ± 25.29 mg/dl, for men and women respectively.

The evidence available from various epidemiological, pathological, clinical and other exploratory studies throws light on the fact that IHD is widely prevalent in the Indian sub-continent and that hyper lipidemia plays a very significant role in increasing the susceptibility of an individual to IHD. SC and STG levels are influenced by a host of other risk factors which act by increasing the blood lipid levels. Some of the more important risk factors are smoking, hypertension, heredity, obesity and coronary prone behaviour.

1.2 Risk Factors other than Diet.

The coronary risk factors are those abnormalities demonstrable in persons free of clinical coronary heart disease and known to be associated with significantly increased risk of developing the disease in subsequent years (Stamler *et al* 1966). They include hyperlipidemia, hypertension, cigarette smoking, over-weight, positive family history of premature vascular disease (e.g., onset before age 60) etc. A habitual dietary pattern high in total calories, total fats, saturated fats, cholesterol, refined carbohydrates and salt is a major coronary risk factor, since it can be of key importance in contributing to the development of hyperlipidemia, obesity, hypertension etc. All these risk factors have been implicated in the pathogenesis of atherosclerotic diseases and permit assessment of susceptibility or proneness to coronary disease on a probability basis.

1.2.1. Family History

The role of heredity and environment is of prime importance in the understanding of the nature of chronic diseases. The Genetic mechanism must be known for a better understanding of pathogenesis. Knowledge of the way in which a genetic predis-

position toward a clinical disease interacts with specific environmental forces may indicate the appropriate means for its control. Study of the distributions of blood pressure levels in a population, and of the degree of resemblance within families and kindreds, presents a good opportunity to evaluate these relationships.

John *et al* (1965) concluded from their study that parents with high levels tend to have children with relatively high levels, while low levels in parents are likewise reflected in low levels in their offsprings.

Skyring *et al* (1963) reported about a pilot study on some epidemiological and familial aspects of CHD. The results revealed that the children of the proven and probable coronary deaths had higher mean cholesterol levels than the controls. However, the differences were statistically significant only in the case of male children.

Significant correlations between cholesterol levels of either father or mother and their children and between siblings were reported by Adlersberg and Schaefer (1959). Schaefer *et al* (1958) had shown significantly higher correlations for mother – child, than for father – child comparisons, though no sex linkage was found.

Explaining about hereditary aspects of CHD, Epstein (1964) gave evidence that CHD, did tend to aggregate in families. Families share their environment as well as their genes. Disease of the coronary arteries was twice as common a cause of death in the fathers of patients as in the fathers of the control subjects (37 vs 19 per cent). Nine of the siblings of patients died of CHD, as opposed to one per cent of the siblings of controls. The results suggested that the familial factor is, infact, very important. The data further indicated that a positive family history carried appreciable weight.

Epstein quoted another investigation in which it was made clear that when both grand parents had CHD, 21.2 per cent of their sons were similarly affected; when neither grand parent had

the disease, only 4.1 per cent of the sons were reported to have the same condition. It is of considerable interest that the disease became manifest in the sons, on an average 20 years earlier than in their fathers, which suggested that environmental changes over the past few decades might have tended to bring a genetic predisposition increasingly into the open.

A summary of the correlation coefficients for SC values in parents and their children indicated a regular, although not strong, tendency for parents and children to resemble each other over the whole range of distribution rather than just in the upper range, since their regressions could be shown to be essentially linear. The association between CHD and elevated levels of SC and BP makes it likely that the determinants of these particular two variables are an integral part of an atherosclerotic constitution. Long term studies of families help to understand the true extent of familial aggregations of CHD.

Osborne and Adlersberg (1958 and 1959) made a study of monozygotic and dizygotic healthy adult twins and showed the importance of both genetic and environmental factors in the regulation of serum lipid levels. McDonough *et al* (1962) also concluded from their study of 56 monozygotic and dizygotic twins, that factors affecting the variation in cholesterol levels were both environmental and genetic.

A genetically determined bio–chemical defect may be there, the expression of which is modified by environmental or other genetic factors (Johnson *et al*, 1965). Avid Heiberg (1974) studied the heritability of serum lipids and lipoproteins in twins. He concluded stating that cholesterol values are genetically influenced and that 56-84 percent of this variation is genetically determined. A substantial genetic influence was also apparent on other serum lipoproteins and lipids in this series of young healthy twins. Thus this study further emphasises the importance of genetic factors in determinining the concentration of serum lipids and lipoproteins.

Children whose parents, especially the fathers have had a myocardial infarction before age 50, showed hyper cholesterole-

mia and hyper lipidemia (Glueck *et al* 1974; Blumenthal *et al* 1985; Hennekens *et al* 1976; Levine *et al* 1978; Rissanen and Nikkila 1977 and Anderson *et al* 1981). Nikkila and Aro (1973) reported that there was significant clustering of hyperlipidemia and that there is a familial trait of hyperlipidemia in 1/3 of patients with Ischemic Heart Disease.

An Indian study (Vajpayee *et al*, 1981) reported about the serum lipids in first degree relatives of patients of IHD. First degree relatives of 57 patients of documented IHD patients in the age group of 21–40 were studied and compared with 30 healthy invidividuals. There was a significant difference in serum cholesterol levels between the relatives of IHD patients and controls (P<0.01), while serum triglyceride levels in the two groups were not significantly different. Hypercholesterolemia was observed in 52.5 per cent of smoker and 48.7 per cent of non-smoker relatives of IHD patients, while it was observed only in 6.66 and zero per cent respectively of control individuals.

Kare Berg (1989) stated that use of the methods of quantitative genetics indicates a strong effect of genes on several indices known or believed to be related to susceptibility or resistance to CHD. However, with the exception of LP(a) Lipoprotein level, genetic factors explained only part of the quantitative variations, leaving ample space for risk factor modification by manipulation of life style or diet.

The studies on family history so far reviewed point to the important role family history plays in IHD and hyperlipidemia. Several parent–child studies have clearly shown relationships between parents and children in serum cholesterol and blood pressure levels. Studies on twins also confirm the genetic influence on serum cholesterol levels. Nevertheless, since families share their environment as well as their genes, it is difficult to separate the environmental influences from genetic influences. All the same, it cannot be denied that both hereditary as well as environmental influences operate in consonance. These observations need to be examined in an Indian context.

1.2.2 Cigarette smoking

Numerous studies have established smoking to be a maior coronary risk factor. Not only is smoking associated with a high incidence of myocardial infarction but also with increased risk of sudden death especially in young and middle aged. It was estimated from data in the prospective study in Framingham that, if cigarette smoking could be completely abolished, the appalling toll of deaths and illness from CHD might be reduced by almost one half (Schettler and Boyd, 1969).

Incidence of myocardial infarction was nearly 3 times greater for cigarette smokers (20+) than in non-smokers. Men aged 40–49 who smoked 40 cigarettes a day had 5 times the risk of dying from IHD compared with non-smokers (Hammond *et al*, 1972). Throughout the 45–64 age range the mortality from CHD among hypertensive smokers was approximately 9–10 times as that of non-hypertensive smokers; and the CHD death rate of smokers with abnormal Electrocardiogram was almost three times that of non-smokers with abnormal electrocardiogram. In a review of coronary occlusion occurring under the age of 50, it was found that within a group of 133 partients, 89 per cent smoked for more than 20 years. Among them 63 per cent died within one hour after onset (Schettler and Boyd, 1969). Absorbed nicotine stimulates the release of norepinephrine and epinephrine. Catecholamines can also be released by nicotine from extra-adrenal chromaffin cells found in cardiac and other tissues. The catecholamines cause the mobilization of free fatty acids from adipose tissues. A rapid rise of serum free fatty acids were shown as an effect of inhaled nicotine.

Nicotine causes an increased sensitivity of platelets to aggregating agents. Smoking a single cigarette increased the response of the smoker's platelets to ADP. Prolonged exposure to carbon monoxide accelerated the development of atheroma in rabbits because of intimal accretion of platelets and fibrin and intramural lipid accumulation. The effects of smoking on platelet survival, platelet aggregation and possibly on vessel walls

may be relevant to the fact that the subjects who give up smoking showed a reduced incidence of clinical complications of vascular disease (Schettler and Weizel, 1974).

Kannel (1977b) stated that cigarette smoking powerfully and independently contributes to the devclopment of lethal coronary attacks, particularly in the young male coronary candidate. The data from Framingham study is presented in Table 6.

Table 6: Twenty Two Year Incidence of Coronary Heart Disease According to Cigarette Smoking Habit (22 year Follow-up Framingham Study) in Men Aged 30 to 39

	Population at Risk	CHD Cases	22-year Incidence per 100
Men			
Non–Smokers	214	17	72.4
Cigarette Smokers	585	99	183.4

At older ages the impact is less pronounced. The benefits of giving up the smoking habit at an early age are noteworthy. The risk is reduced to half that of those who continue to smoke. Thus, this is a vital consideration in the preventive management of the young coronary candidate.

A smoker's risk of developing atherosclerosis can be related to the carboxy haemoglobin (CoHb) levels. Carbon monoxide (Co) is present in tobacco smoke in concentrations of about 4 per cent. It readily diffuses through the lungs and is avidly taken up by haemoglobin to form stable pigment CoHb. The per cent CoHb saturation is determined by a number of factors, including tobacco consumption, concentration of co in tobacco smoke, depth of inhaling, Co pulmonary transfer factor and atmospheric Co levels. Atmosphere Co very rarely results in CoHb levels above

2.5 per cent, Values above this are usually the result of smoking and may reach 15 per cent in heavy smokers. A CoHb value provides information on tobacco smoke absorption, on depth of inhaling as well as on recent tobacco consumption (Waldet all, 1973).

High carboxy haemoglobin levels were found to be strongly associated with an increased frequency of atherosclerosis. If cigarettes are inhaled more deeply, CoHb level goes up. The vascular changes may come about as a result of the decreased oxygen supply (Thiele, 1980),

The harmful effects of smoking are likely to be related to tobacco itself as well as to the products of burning of wrapping material. In India, smoking of tobacco is mostly in the form of cigarette or beedi and a study showed that rise in carboxy haemoglobin level was somewhat more after smoking beedi than after cigarette. This difference is possibly related to lesser porosity and poor combustability of beedi leaf compared to cigarette paper (Gupta *et al* 1980).

Hjermann *et al* (1981) studied the effect of smoking intervention and reported that significant reduction in blood lipid levels and in the incidence of coronary disease is possible with smoking and dietary intervention.

Sinha *et al* (1985) carried out a study to investigate the effect of smoking on blood pressure, body weight, electrocardiogram and serum cholesterol in young hypertensive smokers (a) normotensive smokers (b) and normotensive non – smokers controls, (c). Effect of smoking was observed in the first two groups while the last was kept as control. Smoking caused a rise in mean systolic and diastolic blood pressure of both hyper and normotensives as compared to controls. The rise was always greater in hypertensives than normotensives. Smoking also contributed to significant increase in serum cholesterol in hypertensive and normotensive volunteers. The serum cholesterol rose by 75.20 mg per cent in hypertensive smokers and 53.7 mg per cent in normotensive smokers. Smoking produced various clinico–biochemical changes. The rise in blood

pressure after smoking is mediated by sympathetic stimulation, leading to release of catecholamines. Though many of the changes were more marked in hypertensives than normotensives suggesting an increased sensitivity in hypertensives, these multi-faceted effects of smoking lead to various cardiovascular complications. in the long run, more so in hypertensives.

The biochemical profile of smokers and non-smokers was studied by Vijayalakshmi and Lakshmi (1985). Forty volunteers who had a previous incidence of myocardial infarction were chosen. Among these 20 were smokers and 20 were non-smokers. Another thirty volunteers, 15 smokers and 15 non-smokers who had no history of myocardial infarction were also chosen for the study, making a total of seventy altogether. All the volunteers were closely matched for activity, dietary habits and socio-economic level and the age ranged from 30 - 60 years.

The most common age at which both the smokers and non-smokers had the heart attack was between 40 and 50 years. However, those who smoked had a tendency to become heart patients much earlier (30 - 40 years) than non-smokers.

The. estimated serum cholesterol and serum triglyceride levels in healthy controls and heart patients are given in Table 7.

Table 7 : Mean Serum Cholesterol and Serum Triglyceride Levels in Healthy Controls and Heart Patients

Healthy Controls (mg/100ml)				Heart Patients (mg/100 ml)			
Smokers		Non-Smokers		Smokers		Non-Smokers	
SC	STG	SC	STG	SC	STG	SC	STG
225.9 ±20.56	126.3 ±18.3	202.4 ±23.7	110.3 ±19.0	279.2 ±13.74	163.3 ±24.9	255.4 ±9.38	148.6 ±13.29

The serum cholesterol and serum triglyceride levels of smokers were higher in both the groups. Compared with the values of healthy, non-smokers, all the values were more and these differences were statistically significant at one per cent level. The acceptable levels for cholesterol were given as 140-250 mg/100 ml and for triglyceride it was given as 40-160 mg/100 ml.

The mean systolic pressure was low in healthy controls (smokers 122 ± 13.5, non-smokers 120 ± 13.69) compared to heart patients (smokers-133. ± 19.4, non smokers 125 ± 13.92) irrespective of the smoking habits. The diastolic pressure of healthy controls, both smokers and non-smokers, was significantly lower (smoker-88.2 ± 6.4, non-smokers-80 ± 5.8) when compared to the levels registered by heart patients (smoker - 95.2 ± 6.8, non-smokers - 92. ± 6.5). The authors conclude saying that myocardial infarction is taking a very high toll irrespective of the age. Smokers were more susceptible to myocardial infarction at an early age than non-smokers.

The studies on smoking reviewed above, leave little doubt about its role in CHD incidence. There are clear indications that increased smoking is closely related to atherosclerosis. Serum lipid levels (serum cholesterol and serum triglycerides) were much lower in the normotensive non-smokers. Thus, smoking constitutes an important risk factor that is associated with elevated serum cholesterol and triglyceride levels.

1 2.3 Coronary Prone Behaviour

The influence of the mind on matter has been for long a subject of debate. With the development of the disciplines of psychology, psychiatry and psychosomatic medicine, the increasing role of the mind on body processes became common knowledge. Interestingly, the brain is the seat of control of both the behaviour and the biology in man. No wonder, the mind has a significant sway over the heart.

What is the relevance of considering psycho-social factors in contributing to the risk of CHD? Evidence, indirect and direct, retrospective and prospective, has been growing in the

last two or three decades. Literature on the association of social and psychological variables with CHD has been appropriately reviewed elsewhere (Caffrey, 1967; Jenkins, 1971 and 1976; Leibowitz, 1970).

Questions have been raised on the causes of elevation of cholesterol levels and increased incidence of IHD among Japanese Americans compared to lower rates of IHD among the Japanese in Japan, despite higher intake of saturated fat and smoking of the latter (Marmot and Syme, 1976). IHD rates are up among the city dwelling Israeli Arabs compared to their non-city dwelling nomadic counterparts (Mc Michael, 1979). Maoris who migrated to New zealand had a much higher rate of IHD, compared to those who remained behind, IHD was rare among the native Maoris, despite their higher dietary saturated fat intake (Eaglehole *et al*, 1980). Rosenman (1983) has quoted many studies of similar nature to show that the customary non-psychological factors do not fully explain the variations in incidence of IHD, hinting at the important role of psycho-social factors. Quoting Keys *et al* (1972) Rosenman adds that even when full account is taken of all conventional risk factors, they fail to account for nearly half the numerical incidence of IHD in prospective studies.

The studies mentioned above go to show that increased rates of CHD seem to be associated with adjusting to new environments, changed situations in jobs, moving upwards in social status and the resulting stress (Kaplan *et al.*, 1971; Medalie *etal*, 1973; Syme *et al*, 1964 & 1965). These factors which appear to act as precipitating agents, seem to lie in psycho-social variables (Jenkins, 1971). A psycho-social variable called the Type-A Behaviour Pattern seems to be the common denominator for the interaction of these variables.

There is strong evidence to support the relationship of Type A Behaviour Pattern (TABP) to IHD. Individuals with Type A Behaviour appear to have an enhaneed rate of intimal damage (Rosenman, 1983). The type A Behaviour Pattern has been described by Friedman and Rosenman (1959) to be characterised by intense ambition, competitive drive, constant pre-occupation

with occupational deadlines and a keen sense of time urgency. The converse of this behaviour called Type-B Behaviour is said to be characterised by a more relaxed, unhurried, more easily satisfied approach with lesser time urgency and lesser competitive hostility. Probably Type A Behaviour could be defined in terms of a limited set of descriptive characteristics namely, competitive achievement striving, exaggerated sense of time urgency, aggresiveness and hostility. In clinical practice, the designation of a person as Type A or Type B, depends upon the summation of the number of Type A characteristics and their intensity (Jenkins, 1975).

Several new assessment procedures and defining correlates of TABP (Type A Behaviour Pattern) have given Type A and Type B researches an array of psychometric questionnaries. Some of them are the Adjective check list (Gough and Heilburn, 1980). Thurstone Temperament Schedule (Thurstone, 1949), Eysenck Personality Inventory (Eysenck and Eysenck, 19 68), State Trait Anxiety Inventory (Spielberger *et al*, 1970), work Environment Scale (Insel and Moos, 1970), California Psychological Inventory (Gough, 1956), The MMPI (Dahlstrom and Welsch, 1960), the 16 PF (cattell, *et al*, 1953) and Barratt impulsive scale (Barrat, 1965). Despite the availability of these scales, the hurry, poor insight and inadequate self appraisal of many Type As are thought to lead to inaccuracy that limits the ability of these questionnaries to serve as measures of TABP (Roseman *et al*, 1976; Rosenman, 1978).

Of the questionnaries to measure TABP, a good and well studied one is the Jenkins Activity Survey (JAS) which is being increasingly used (Jenkins *et al*, 1979). The JAS provides a composite Type A scale and three sub-scales, viz., Speed and Impatience, Job Involvement aud Hard Driving derived through independent factor analysis. The TABP assessed through the JAS, has been shown to be significantly related to CHD prevalence in many populations. Also, JAS scores for TABP were predictive of new cases of CHD (Jenkins *et al*, 1974), risk of *reinfarction* in the Western Collaborative Group Study (Jenkins, 1976) and degree of basic coronary atherosclerosis (Zyzanski *et al*, 1976).

The JAS is as good as the standardised structured interview (Kittel *et al*, 1978). The present form of the test questionnaire distinguishes at high levels of statistical significance, those judged not to manifest the pattern and those who manifest the pattern. The tests capacity for categorisation of invividuals is said to be promising. Moreover, a series of validity studies using biological and medical criteria have been carried out (Jenkins *et al*, 1967).

It is more than two decades now since a significant association between TABP and CHD was reported (Friedman and Rosenman, 1959; Rosenman and Friedman, 1961). Several others have confirmed this association subsequently (e.g., Feinleib *et al*, 1978; Rosenman and Chesney, 1980). Higher rates of coronary artery disease in the USA compared to Europe was explained in terms of TABP by Keys *et al*, 1972. Kozaravic *et al*, (1976) attributed differences in CHD between the Framingham men and men in Yugoslavia as due to TABP. More emphatically, the Western Collaborative Group Study (W.C.G.S.) which was a prospective epidemiological study of 3154 men aged 39–59 years has confirmed the association between TABP and CHD (Rosenman *et al*, 1964). There are several other studies, which have indicated a positive association between Type A Behaviour and coronary diseases (viz., caffrey *et al*, 1969; Keith *et al*, 1965; Jenkins *et al*, 1971; Renigsberg *et al*, 1974; Shekelle *et al*, 1976; Glass *et al*, 1977; Zyzanski *et al*, 1979). Further in an 8 4 year follow up study, Rosenman *et al*, (1975) reported that men who were classified as Type A at intake were 2.37 times likely to develop CHD over the follow up period compared to Type B subjects Further, when this value was adjusted to remove effects of other standard risk factors, the values got reduced only to 1.97 (Rosenman *et al*, 1976). These data show that substantial risk is associated directly with the possession of Type A Behaviour pattern.

There is a sizable literature concerning the relationship between environmental stressors and serum cholesterol response. Apart from animal studies (e.g., Friedman, Byers and Brown, 1967) several studies have shown association between psychological states and serum concentrations of cholesterol in human

beings (e.g., Mann and Whit, 1953; Rahe, Rubin and Arthur, 1974; Rahe, Rubin, Gunderson and Arthur, 1971). These studies showed elevated serum cholesterol levels when the individuals were overburdened by environmental demands.

Early in research on TABP, a link was observed between TABP and serum cholesterol. Significant rises in cholesterol levels were noted among accountants with deadlines to meet and experiencing time pressure and occupational annoyance (Friedman *et al*, 1958; Rosenman and Friedman, 1974). Average serum cholesterol tended to be higher in Type A than Type B subjects belonging to both the sexes (Friedman and Rosenman, 1958; Friedman, Rosenman and Byers; 1964; Rosenman and Friedman, 1961 and 1963). In another study of cholesterol levels of Type A and Type B students, the mean level of cholesterol of Type As (198.3 mg per cent) exceeded significantly that of Type B (168 mg per cent) students (Glass, 1977).

Type As also exhibit higher fasting and post prandial triglyceride levels than do their counterparts (Rosenman and Friedman, 1963), along with sludging of red blood cells in the bulbar conjuctival blood vessels for many hours after ingestion of a meal rich in either saturated or unsaturated triglycerides (Rosenman and Friedman, 1954). The association of psychological states with serum–lipoprotein levels has been reviewed in detail elsewhere (Jenkins *et al*, 1969).

Blumenthal *et al* (1975) found that 82 per cent of 72 patients who had 75 per cent of narrowiug of coronary artery turned out to have been classified as Type A, whereas 63 per cent of patients without significant disease were classified as Type B clinically. These studiese show that pattern A increases the risk of CHD at least in part through an association with the atherosclerotic process.

Catecholamines probably play an importanl role in mediating relationship between TABP and both coronary atherosolerosis and CHD incidence. Raab (1953) recognised that adrenergic responses were enhanced in TABP individiduals, compared to more relaxed Type B persons (Raab, 1960). In fact it has been

found that subjects with CHD exhibit enhanced catecholamine discharge in relation to emotional and other stress and during physical exertion (Keys *et al*, 1971; Nestle, Verghese and Lovell, 1967; Voudoukis, 1971). Such enhanced discharge is associated with aggressiveness, competitive drive, anger and time urgency which are major TABP facets and this has been found in atherosclerotic subjects (Brozek *et al*, 1966; Fimadjian *et al*, 1958). Incresed nor-epinephrine excretion by Type A subjects has been reported in work settings (Friedman *et al*, 1960) and in competitive situations (Dembroski *et al*, 1978; Friedman *et al*, 1975; simpson *et al*, 1974). Further, Type As compared to Type Bs have been found to exhibit a greater rise of heart rate, systolic and/or diastolic blood pressure and catecholamine secretion during the structured interviews, reaction time tests, exposure to noise and uncontrollable aversive factors and psychomotor performance tests (Dembroski *et al*, 1978; Friedman *et al*, 1975; Lovallo and Pishkin, 1978).

Little correlation of TABP has been found with age except for lower prevalence at younger ages (Rosenman *et al*, 1964; Shekelle, Scholnberger and Stamler, 1976). However, in an Indian study (Jamuna and Sujatha Ramamurthi, 1984) there was a relationship between age and JAS scores indicating an increase in JAS scores with age. The scores were highest in the middle years. The TABP shows *modest* correlations with occupational status (Rosenman and chesney, 1980; Waldron *et al*, 1982) and career advancement and achievement in both sexes (Waldron *et al*, 1978). An Indian study (Ramamurti, Jamuna and Sujatha Ramamurthi, 1984) showed that the middle aged executives had higher JAS scores compared to non-executives.

In the foregoing, some of the more significant studies on coronary prone behaviour have been reviewed. Several measures are available for assessment of coronary prone behaviour. Chief among them being the srtructured interview and the Jenkins Activity Survey (JAS). The TABP was significantly related to the occurence of IHD. This fact has been well documented through both prospective and retrospective studies. Also, TABP has been shown to be related to cholesterol, triglycerides, catecholamines and to angiographic findings. In essence, the reviewed literature

points to a firm implication of TABP in influencing levels of cholesterol and in the occurence of IHD. Most of the evidence has come from studies carried out in the U. S. A. and Europe. There are hardly any studies in India relating TABP to either hyperlipidemia or incidence of IHD. Thus, there is a need to examine this relationship in an Indian setting.

1.2.4 Hypertension

Elevated blood pressure is a major public health problem of our age. It increases the risk of several major cardiovascular diseases and premature death. Williams *et al*, (1983) reported that as many as 65 per cent of the elderly population, who had their first stroke, had a previous history of hypertension. High blood pressure had for long been identified as one of the three major CHD risk factors (Tillotson *et al*, 1984).

The clinical manifestations of IHD have been frequently studied in relation to high blood pressure. Numerous retrospective studies have been made by various authors, of the antecedent hypertension in patients with cardiac infarction; these have shown that in both males and females a large proportion (varying in different series from 40–70 per cent) were hypertensives (Wilson, 1969). In the Framingham studies, over a six year period, a direct correlation between hypertension and the incidence of CHD was observed. In men aged 40–50, a 2.6 fold increase in CHD was observed when hypertension was present and in women of the same age group, it was a six fold increase (Kannel, 1961).

Blood pressure and atheroma increase with age and it is difficult to separate these factors. Epidemiological studies provide clear-cut evidence that hypertension is an important predisposing cause in CHD. Arterial hypertension increases vessel wall permeability to blood constituents, particularly proteins and lipids, thereby producing extracellular deposits which form the basis of degenerative arterial changes (Wilson 1969).

In the International Atherosclerosis Project (IAP) 23,000 aortas and sets of Coronary arteries were studied, including some 2000 hypertensive subjects The hypertensive subjects were compared wirh normotensives with regard to the extent of fatty streaks and raised lesions (fibrous plaques, complicated and calcified lesions) separately and combined in the aorta and the three coronary arteries. There were significant differences in the extent of fatty streaks in the abdominal aorta between hypertensive and non–hypertensives, particularly in the younger men (25–44 years). At all ages there were differences in raised lesions between the two groups. In the coronary arteries, both fatty streaks and raised lesions were significantly more frequent in hypertensives at ages 25 to 54 in men and women. Hypertensive subjects had a higher mortality rate at younger ages than normotensives and this was proportional to the severity of the hypertension. Over all, the IAP showed that hypertension accelerated the progress of atherosclerosis, particularly with regard to the raised lesions (Robertson and Strong, 1968). The age–adjusted annual incidence cases per 10,000 persons (Table 12) increased with increased systolic pressure in the 18 years follow up of the Framingham study (Kannel, 1977). Hypertension is associated with an excess rate of development of every major clinical manifestation of CHD.

Table 12 : 22 Year incidence of Coronary Heart Disease According to Hypertensive Status, Framingham Study, Men Aged 30–39.

	Population at risk	CHD	22 Year Incidence per 1,000 Crude*	22 Year Incidence per 1,000 Corrected**
Men				
Normotensive	497	54	108.7	114.2
Borderline	180	27	150.0	158.7
Hypertensive	155	37	238.7	271.1

* Crude = No. of cases in 22 years – No. at Risk
** Corrected = No. of cases – Person–years at Risk x 22 years.

If control of hypertension is instituted early enough in life, before target organ involvement and sustained for long periods, it could rationally be expected to slow the accelerated pace of atherogenesis which accompanies hypertension (Kannel, 1977[2]).

The incidence of hypertension varies from country to country. In the U.S.A., Judd *et al* (1983) estimated that 33 million people suffer from hypertension, while Whitney and Cataldo (1983) stated that 60 million Americans are affected by hypertension. However, the incidence rates for thc developing countries like India are much lower than in the Western countries Bhatia (1978) reported that nearly 15 per cent of urban and 4 per cent of rural Indian populations are estimated to be hypertensives.

Sapru (1984), giving a lowest estimate of the prevalence of CVD in India, stated that 15.8 million patients were suffering from hypertension and 330 millions were at risk. The prevalence of hypertension in different population segments in India varied from 3.57 per cent to 6.43 per cent in the age group 20 years and above, with an average value of 4.8 per cent. An estimate of new cases of hypertension likely to appear every year during the present decade worked out to be 720,000.

At present hypertension is defined as a systolic blood pressure >160 mm Hg and a diastolic blood pressure >95 mm Hg (WHG, 1978). In young adults mild hypertension has been arbitrarily defined at pressures exceeding 140/90 mm Hg (at or near the 90th percentile) and definite hypertension at pressures more than 160/95 mm Hg (at or near the 95th percentile). In recent years, it has been shown that an elevation of blood pressure above 149/90 mm Hg to be above the normal range and that a blood pressure below this level greatly reduces the risk of cardiac faiure and other complications (Robertson, 1978).

The differences in blood pressure levels as well as the incidence of hypertension in population is believed to be due to genetic and environmental factors. But in populations of a

similar environment, the differences in blood pressure may be attributed to genetic factors (Prior, 1978 and Marmot, 1984). Apart from race, age, sex and somatic factors, blood pressure is also known to be influenced by many others such as, socio – economic status, cultural and occupational factors (Gupta *et al*, 1979; Indrayan *et al*, 1972; Malhotra and Ganguly, 1976; Marmot, 1984; Sambasiva Rao, 1984). Also, blood pressure of an invididual is acquired as he acquires height (Robertson, 1978). Psychological factors, particularly stress is an important contributor to blood pressure (Chandra Patel, 1983),

Age and Sex ; Age seems to have an effect on blood pressure. Blood pressure rises with increase in age due to the structural changes in the arteries. As age increases, the arterises get hardened due to mineralization and thus bocome resistant to blood flow. (Best and Taylor, 1984; Garden and Eugene, 1983),

Does gender influence blood pressure? Females had slightly lower values of blood pressure than males. This was attributed to the protective nature of the female sex hormones and to their smaller body stature (Indrayan *et al*, 1972b; Srivastava *et al*, 1977; and Gupta *et al*, 1979).

Recent views of studies of blood preasure in different cultural groups are in general agreement that blood pressures of adults tend to be higher in Westernized societies than in unacculturated traditional societies. In Westernized societies, blood pressure tends to increase with age. In contrast, in many traditional societies, there is little or no relationship between blood pressure and age for adults. The protective effects of very low salt consumption in some groups and differences in body weight appear to contribute to cross cultural variation in blood pressure (Waldron *et al* 1982).

The foregoing studies on hypertension suggest that hypertension is a major risk factor contributing to CHD. In view of the fact that both atheroma and hypertension increase with age, it is difficult to separate their independent roles. An association

between hypertension and raised cholesterol (lipid) levels has been presented. Also there are some studies that associate elevated blood pressure with stress and with life styles characterized by an urbanised money economy.

1.2.5 Obesity

The bulge around the belly has been for long a matter of concern for man and woman alike. Obesity is a major malnutrition problem. Also, it is one of the more important but less understood areas in the science of nutrition. Many know what it is, but few know what to do about it.

Overweight results from an unbalanced energy budget. The overweight person consumes more food energy (K. calories) than he expends and has banked the surplus in his fat cells.

Obesity is associated with the presence of several diseases like hypertension, CHD, diabetes mellitus, arthritis etc. Though obesity is a weak and inconsistent predictor of CHD by itself, it predisposes to the development of hypertension and diabetes, which are themselves important risk factors (Davidson and Passmore, 1975). Excessive weight of the body or obesity increases the stress on the cardiovascular system. Thus, obesity can be considered as a risk factor for developing coronary artery disease. In some instances, cardiac enlargement and congestive heart failure are the result of obesity (Thiele, 1980).

Obesity may be classified as either exogenous, meaning that it is caused by excessive energy intake or endogenous, meaning that there is an inherent metabolic problem that promoted the obesity. Obesity resulting from an excess intake of calories is the most common. Physiologically, obesity can be categorised on the basis of adipose cellularity. Fat cells may increase in number (hyperplasia) or size (hypertrophy). Adipose tissue number increases during the first few years of life and again during the period between 9 and 13 years of age [Salans *et ol*, 1978].

Obesity promotes CHD, by enhancing lipidemia i.e overproduction of cholesterol and triglyceride. Keys [1970] analysed the relationship of relative weight and skin fold thickness to the 5 years incidence of CHD in 11,400 men aged 40–59. Multivariate analysis of the data showed that neither relative weight, nor obesity was in itself a statistically significant risk in relation to future CHD, when age, blood pressure, serum cholesterol [SC] and smoking were comparable. But, as expected, relative weight and fatness correlated with BP and SC.

It may be true, as the findings suggest, that obesity itself does not not necessarily increase the risk of CHD. Though overweight may not be an independent risk factor, there are many compelling grounds for weight reduction. In the first place, a diseased heart should not be subjected to the unnecessary strain of moving an overweight body. Secondly, obesity is associated with the development of hypertension, hyperlipidemia and diabetes, all of which are etiologically important in CHD. Weight reduction should be the first and is often the most successful treatment for these conditions [B.M.J., 1973].

Overweight is generally accompanied by a worsening of the major atherogenic traits. It is associated with an increased risk of angina pectoris [Table 13]. By weight reduction, overweight coronary prone persons can improve their risk profile. Weight reduction would seem the first important measure to be considered for hypertension, hyperlipidemia and impaired carbohydrate tolerance in the young coronary candidate [Kannel, 1977b].

Table 13: 22 Year Incidence of Angina Pectoris According to Relative Weight Framingham Study. [Men Aged 30–39]

Relative Weight	Population at Risk	CHD	22 Year Incidence per 1,000
<100	425	27	67.1
100–129	371	35	99.5
>129	31	4	148.6

Opie (1973) explained that obesity can contribute to lipid deposition in the artery by promoting high circulating glucose and insulin concentration and by a high fasting free fatty acid concentration. The latter causes an increased oxygen demand. Exercise could decrease the oxygen demand of myocardium but obese subjects fail to exercise vigorously. Hypoxia, blood pressure and mechanical injury could increase the permeability of the endothelium of the arterial wall to exogenous lipid in the form of cholesterol and lipoprotein. By providing high free fatty acid levels and conditions of hypoxia, obesity can contribute to lipid deposition in the artery. Obesity and hypertension co-exist in many cases [Schettler and Boyd, 1969]. In obesity the very low density lipoprotein levels have been shown to be very high. Therefore through these means, obesity enhances the process of atherosclerosis.

Weight gain and overweight have been shown to contribute to higher SC levels and blood pressure values, but obesity did not always show high levels of SC or B.P. Obesity and its relation to CHD was controversial and some investigators stated that there was no relationship between the two [Gertler et al, 1964; Gofman and Young, 1963]. Angina Pectoris, but not myocardial infarction, was shown to have an association with overweight (Kannel *et al*, 1967).

Substantial drop in cholesterol value with weight loss was reported by Moses (1963). Stone (1972) observed significant correlation between obesity and prevalence of M Predominant hyperlipoproteinemia (Type IV) i.e. 5.6 per cent in slim tertiles and 24.8 percent in obese tertiles ($P<0.001$). With a 1100 calorie diet (reducing) this lipoproteinemia was brought down.

Studying the aerobic capacity, obesity and atherosclerotic risk factors in male adolescents, Fripp *et al*, (1985) stated that in their group, obesity as determined by body mass index, provided the largest explanation of the individual variance on the risk factors studied and that if physical fitness programmes are to be used to reduce the high incidence of risk factors, they must be designed to result in a reduction in body mass index (weight) in order to obtain maximum benefits.

It is now suggested that obesity is an independent risk factor for cardio vascular disease (Simopoulos, 1985). Body weight was the most important determinant of serum cholesterol in the Zutphen study (Kromhout, 1983, Nut. Rev. 1985).

Larsson *et al* (1984) made a large number of anthropometric and other measurements on men and stated that in middle aged men, the distribution of fat deposits may be a better predictor of CHD and death than the degree of obesity. Elevated waist-to-hip circumference ratio was a better predictor of risk for CHD and stroke in men than other indices of obesity (Nut. Rev. 1985).

The prevalence of obesity is high in the west. Its prevalence in developing countries is generally believed to be low, but documented information in this regard is scanty. In India, overnutrition, another profile of malnutrition has not been investigated in much detail. The magnitude of the problem is not known, but hospital experience has shown that a large number of obese, patients need management for their obesity.

Exercise and Increased Activity: Exercise as part of a weight reduction programme has physiologic, psychologic and social advantages. Physiologically, it results in improved cardiovascular functioning, increased caloric expenditure and positive changes in body composition. Psychologically the effects can be profound. Some effects are, reduction in tension and stress levels, better sleep and rest, better attitudes and performance at work etc. Socially, exercise can be the focus of strong positive social reinforcement in the process of weight reduction (Heinselman *et al*, 1970).

Physical Activity and Atherosclerosis: Epidemiological and clinical studies have suggested that physical activity delays the onset or slows the progression of coronary atherosclerosis. Controlling for age, body mass index, alcohol use and smoking. Haskell *et al*, (1979) found that strenuous exercise was associated with higher levels of High Density Lipoprotein Cholesterol (HDLC) in men and women.

Physical activity may reduce the risk of CHD through a beneficial effect on the concentration of plasma total cholesterol and the distribution of cholesterol among lipoprotein fractions (Moore, 1982). Strenuous physical activity as exemplified by running, swimming and tennis increases plasma HDL-C levels (Paul *et al*, 1980). Maureen *et al* (1982) showed that plasma HDL–C was significantly elevated in young women who participated in an intensive, but not moderated exercise regimen.

Quig *et al*, (1983) reported that the men who exercised exhibited significant improvements in cardio–respiratory fitness. The exercising group exhibited significant elevation in plasma total cholesterol with non–significant increase in very low density lipoprotein cholesterol, low density lipoprotein cholesterol and consistent weekly increase in HDL–C with aerobic conditioning.

1.2.6. Multiple Risk Factor Studies.

Many of the studies that have been examined pertain to a single variable or two variables that have been considered in relation to CHD. However, there are a few studies that have examined the simultaneous relationships of many variables. Of particular interest are studies employing the multivariate design. Such an approach is but appropriate since both CHD and hyperlipidemia are of multiple causation. In such instances, if we have to understand the etiology of these conditions, it is necessary to evaluate both the independent and relative contributions of the multiple factors, not only retrospectively but also prospectively.

The large scale prospective epidemiological studies have made it possible to assess the impact of two or more risk factors present in combination. Thus the Framingham study report given by Kannel and Gordon (1973) gave information on risk factors and their relationship to CHD. Kannel and Gordon (1980) stated that using ordinary office procedures (blood pressure, electrocardiogram and a cigarette history) and simple laboratory tests (a casual blood test for sugar and cholesterol) it is possible to estimate risk of a variety of major cardiovascular events over a wide range. Using this information, risk can be estimated detecting those with multiple marginal abnormalities

at high risk as well as those with marked elevations of single risk factors. Based on risk decile, less than 2 per cent of the subsequent CHD cases were found on the lowest decile whereas 25 per cent of the cases for men were found in the highest decile.

No major innovations are needed to identify coronary conditions or to establish their risk. However, we have to develop skills in motivating changes in behaviour to control risk factors such as cigarette habit, over weight, hypertension and sedantary living.

Prospective epidemiologic studies provide a valuable approach for determining many of the individual and environmental factors which precede the emergence of CHD and which may raise the occurrence and recurrence. The Western collaborative Group Study (WCGS), initiated in 1960–61 on 3524 men employed in San Francisco, is a continuing prospective epidemiologic investigation of this type. The subjects were followed up for 4½ years. Clinical CHD occurred in 133 initially well men during the period of follow–up. This led to annual incidence of 9.3/1000 men at risk.

With regard to parental history, in 2664 subjects at risk with no parental history, 6.3 was the annual rate of incidence of CHD/1000 men. With presence of parental history (518 subjects) the rate of CHD was 24.5/1000 men. Significant association (P= <0 001) was observed between parental history, cigarette smoking, diastolic blood pressure, serum cholesterol, serum triglyceride and Type A Behaviour Pattern and incidence of CHD in the population studied. In the younger age group (39–49), serum cholesterol was found to be a strong independent predictor of CHD, the strengh of which prevailed even when the influences of beta/alpha lipoprotein ratio and serum triglycerides were limited. An elevated serum triglyceride appear to confer higher risk of CHD, but the effect is pronounced only for men in the higher cholesterol group. It would appear that the maximum CHD risk occurs in the younger subjects when both the serum cholesterol and beta/alpha lipoprotein ratios are elevated, but occurs in older subjects when either fraction is elevated, as herein defined. In the older age groups, bivariate analysis of CHD rates by the serum cholesterol and triglycerides indicates

that higher serum cholesterol confers added risk in association with either low or high serum tryglyceride, but triglyceride appears clearly to be the stronger predictor of the two factors.

Cholesterol and diastolic blood pressure (DBP) are both strongly related to CHD rate, especially in the younger age group, with cholesterol and DBP, both elevated. DBP shows a highly signifficant independent relationship with CHD in both age groups.

Bordia and Arora (1974) conducted a comparative study on predisposing factors of coronary artery disease (CAD) in rural and urban population. There was an increased incidence of overweight, hyper cholesterolemia and sedantary habits in patients who were living in cities as compared to those living in villages. Smoking habit was very high in villagers and this might have contributed as a very important risk factor to CHD. Various risk factors have been given but the contribution of each variable has not beengiven in this study.

The few studies mentioned above are characterised by the simultaneous consideration of several factors, an approach that attempts at unravelling the intricate interaction among the independent variables and their contribution to the dependent variable. The studies have made salient their relative role. It is studies of this sort that is the need of the hour in the Indian setting. The Indian scenario presents a some what different cultural context and life style from the west and an examinatton of the relative role of the risk factors in contributing to lipidemia in India would perhaps be an useful enterprise,

1.3 Diet in Relation to Blood Lipids

"To a man's heart is through his stomach" is an adage that is perhaps as literally true as by allegory. The health of the heart largely depends on what you eat. To eat or not to eat, what to eat and what not to eat are matters that one has to mind According to Shaper (1972), dietary factors are fundamental to the development of severe atherosclerosis.

The attention nutritionists and physicians have given to the possibility of contributing, by the diet, to the prevention of atherosclerosis and its consequences, has arisen from two aspects of respective information:

— that, deriving from studies on experimental atherosclerosis;

— and that, deriving from anatomopathological, clinical, epidemiological and experimental studies carried out on populations of different countries.

It has been shown by research on experimental atherosclerosis, that by confluence of various exogenous and endogenous factors, hypercholesterolemia, and atherosclerotic lesions are produced. These regress along with hypercholesterolemia if the atherogenic diet is replaced with a normal lipid diet.

1.3.1 Total Calories

Epidemiology teaches us that a rich diet is involved in the pathogenesis of cardio vascular diseases and contributes to the development of obesity, hypertriglyceridemia, hyperglycemia– all implicated as risk factors. A correction of these biochemical parameters by arranging the total calorie intake in order to reach and maintain an ideal body weight is of prime importance in a therapeutic or preventive regimen. In some countries the different incidence of coronary diseases seems to depend more on the total calorie intake, than on specific factors. Yet the effect is not well defined. It seems to depend on energy expenditure and also on individual differences in food utilization. Weight reduction frequently lowers elevated serumlipids (Angelico, 1975).

An interesting relationship between calorie consumption and plasma lipid levels was pointed out by Hartman, (1974). He put forward the hypothesis that the increase of serum lipids with age in affluent societies is mainly due to a parallel increase in body weight. In men, the average increase of body weight between 20 and 40 years is about 12 kg and in the following 20 years only about 2 kg. Total serum lipids show a steep increase between the age of 20 and 40 in men, exactly paralleling the

weight curve. Calorie dependent hyperlipidemias (Type IV) become manifest in men between the age of 20 and 40, that is. during the greatest increase of excess body weight. Dietary measures, therefore, are basic for management of the hyperlipidemias and calorie restriction appears to be paramount.

Mancini (1974) demonstrated changes in plasma lipid concentrations and in body weight in obese patients during total fasting and low calorie intake. During the hypocalorie diet, the hyperlipidemic obese patients showed significant decreases of both serum cholesterol and triglyceride concentrations. With his 15 years of experience on the dietary management of hyperlipidemia and CHD, Lees (1974) showed in his studies that weight loss and a significant reduction in cholesterol and triglyceride levels could be achieved with therapeutic diets.

With regard to the mechanism of the effect of weight reduction on plasma lipid levels, one possible explanation is that there is reduction of the amount of adipose tissue and an increased adipocyte sensitivity to insulin, probably resulting in increased removal of VLDL. In obesity, there is a marked over production of cholesterol (Miettinen, 1971). As soon as negative calorie balance is established, there is rapid early lipid lowering.

Hatch (1974) states that overnutrition, whether it be in terms of intake of total calories, animal fats or cholesterol or simply large meals, exerts stress on the metabolic machinery. The resulting strain or increase in the magnitude of risk factors, differs among individuals according to the relative efficiency of metabolic pathways dictated by polygenic inheritance.

In an epidemiological study (the Zutphen study), during 10 years of follow up, significant correlations were observed between body weight and serum cholesterol. Changes in body weight during 5 and 10 years follow up were strongly positively related to changes in serum cholesterol. Multivariate analysis showed that a change of 1 kg in body weight was accompanied by a change in serum cholesterol of 2 mg/dl (Kromhout, 1983).

Shanmuga Sundaram *et al* (1986) stated that body weight plays an important role in the alterations in major lipoprotein cholesterol contents in response to changes in dietary fat composition. These results supported the hypothesis that body weight is an important determinant of SC in a free living, weight gaining population. Lacomte *et al* (1986) illustrated that low energy diets with high cholesterol (1 gram approximately), increased the plasma cholesterol levels, and the lipoprotein composition was altered. The authors cautioned about the possible adverse affects of slimming diets when associated with high cholesterol intake especially in 'high-responders'.

1.3.2 Dietary fat and Serum Cholesterol Levels and Serum TG Levels.

Epidemiological studies indicate that hyper cholesterolemia is associated with CHD. In population groups whose diets contain fat calories to the extent of 40 per cent, hyper cholesterolemia and CHD are widely prevalent.

In international comparisons, in which correlation between nutrient intake and incidence of CHD was sought, the most consistent correlation was the tendency of susceptible populations to consume latge amounts of triglycerides rich in saturated fatty acids (Fidanza, 1972; Vergroesen, 1972) For eg., a typical North American consumes diet in which fat provides 40 per cent of calorie intake and in which the dominant sources of fat are beef and milk fat. But a Japanese population group, with a low incidence of CHD, was found to consume only 9 per cent of its calories in the form of fat. In the Greek Island of crete, total fat consumption is 39 per cent of calories–nearly as high as in the U. S. A., but it is dominated by olive oil, a fat with a high content of Oleic acid (monounsaturated). In this area, the incidence of IHD is as low as in Japan. Both the Japanese diet and the diet of crete are low in saturated fatty acids, as the diets of crete are low in saturated fatty acids, as the diets of several other areas where the incidence of IHD is low.

The amount and kind of fat in the diet have a considerable effect upon the plasma lipid concentrations. Saturated fat

elevates and polyunsaturated fat decrease plasma cholesterol levels. Monounsaturated acid i.c. oleic acid being the characteristic fatty acid, has a neutral effect and does not in itself either elevate or depress the plasma lipids. The relationship of saturated fatty acids (SFA), cholesterol and poly unsaturated fatty acids (PUSFA) to serum cholesterol levels have been summed up by Keys *et al* (1965) in a regression equation of the following form.

$$C = 1.2 \,(S - P) + 1\,5\,Z$$

In this equation, 'C' is the serum cholesterol concentration 'S' stands for glycerides of saturated fatty acid, 'P' represents glycerides of polyunsaturated fatty acids percentage of total dietary calories and 'Z' the square root of dietary cholesterol in mg/1000 kcal. The implications of the equation are these: as SF in the diet is increased, serum cholesterol will go up; as polyunsaturated fat in the diet is increased, serum cholesterol will go down and the influence of saturated fat is twice as the influence of PUSFA is. The hypocholesterolemic effect of PUSFA is due to palmitic acids. The lesions in the coronary arteries are reversible when the diet is altered to a low fat diet or to one high in polyunsaturated fats. Increased serum cholesterol levels can be reduced by dietary changes, including qualitative and quantitative changes of the dietary fat intake (Nestel *et al*, 1974; Vessby *et al*, 1982; Keys, 1957; Ahres *et al*, 1957; Anderson *et al*, 1957; Beveridge *et al*, 1965).

All animal fats are highly saturated except those derived from marine sources, fish and shell fish. Coconut oil, palmoil and chocolate, perhaps the only saturated vegetable fats commonly consumed in considerable amounts have a hypercholesterolemic effect. Egg yolk lipid and heavily hydrogenated vegeable oils were found to have properties resembling those of ruminant fat. Fish oils were found to resemble the seed oils; and the oils of seed coats (eg. olive oil) were found to have intermediate properties. It became apparent that there was a disparity between the effects of the common seed fats which are generally rich in linoleic acid, and the body and milk fats of ruminants in which saturated fatty acids dominate; substitution

of seed facts (eg. cottonseed, safflower) for ruminant fats consistently induced decreases in cholesterol, the new levels sustained as long as the seed fats were fed (Teymour Dayton, 1975).

The polyunsaturated fatty acid (PUSFA) occuring most frequently in ordinary food products is the essential fatty acid, linoleic. An increase in PUSFA is only half as effective in reducing serum dholesterol as the commensurate decrease in saturated fatty acids. The American heart association recommended fat controlled (quantity and quality) diets, both preventive and therapeutic for the population.

By following such diets wherein the dietary cholesterol is lowered to <300 mg/day, the SFA is decreased (to 4–7 per cent of total calories) and by increasing the polyunsaturated fatty acid content by means of which the P/S ratio is also elevated, substantial and statistically significant decrease in serum lipid levels were observed (Muller, 1973; Gotto and Scott, 1973; Roine, 1972; Gotto, 1974).

In their experiments on rats, Vijayammal *et al* (1982) showed that a high carbohydrate, high protein and high fat diets decreased HDL-C and increased LDL-C and VLDL-C, whereas low protein or fat free diet increased HDL-C. The high protein and high CHO diet decreased adipose tissue LDL (Lipoprotein Lipase) activity. A high fat diet increased the same, Saffola oil caused lower levels of HDL as well as LDL and VLDL-C and higher faecal excretion or sterols and bile acids in comparison to groundnut oil and coconut oil.

Mathur (1983) demonstrated a significant lowering of blood lipid levels by modifying the quality aud quantity of fats in animal and human experiments. Highly significant rise in serum cholesterol level was observed in human subjects when saturated fats like butter and dalda were administered and highly significant fall when unsaturated fats like til oil and mustard oil were given.

In a long term study on humans extending to over one year, the same author recorded a significant rise in serum cholesterol levels with insignificant changes in the fecal excretion of bile acids, when the subjects were on butter and ghee (100g/day) diet. On the contrary, the subjects on mustard oil diet showed not only a highly significant fall in serum cholesterol level but also an increased faecal excretion of bile acids. The author stated that the hypocholesterolemic action observed in the mustard oil group was not significantly different from that seen in the other two groups who were fed equal quantities of mustard oil + dalda and mustard oil + butter or ghee. The author felt that this has practical application since those who are used to ghee or dalda are not prepared to change to oils completely, but can be pursuaded to use ghee for dhal, bread etc., while the main cooking medium for vegetable, fish and other preparations of food can be oil.

Thensen *et al* (1986) treated 14 patients with severe CHD and SC levels of 6–9.5 mmole/L for 3 months with a diet containing 10 per cent of total energy from fat and less than 100 mg cholesterol the serum cholesterol and serum LDL-C were reduced by 33 per cent and 41 per cent respectively, while serum HDL-C and serum TG did not change significantly. After 3 months, patients were asked to maintain a diet as low in fat as possible for long term treatment. After 12 months, a 4–day diet recall showed a mean fat intake of 21.4% (range 7,3–37.8%). On an average serum cholesterol and serum LDL-C were reduced by 14 per cent, and 18 per cent respectively from pretreatment values. Serum TG decreased by 27 per cent and serum HDL-C increased by 18 per cent. The authors felt that this could act as an effective cholesterol lowering diet.

When many investigations were focussing on the effects of saturated and polyunsaturated fatty acids, Mattson and Grundy (1985), included monounsaturated fatty acid also in the comparison and studied the plasma lipids and lipoproteins in man. They compared palm oil (Saturated), high oleic safflower oil (monounsaturated) and high linoleic safflower oil (poly unsaturated) in 20 patients. Both mono and poly unsaturated diets caused statistically significant and equal lowerings of plasma

LDL–C, but the poly diet lowered HDL–C levels more frequently than did the mono diet. Although there was a trend towards reductions in total cholesterol and LDL–C levels by both types of unsaturated fats, the changes were inconsistent. Further more, HDL–C concentrations were low on the saturated fat diet and were unaffected by either the mono or the poly diet. The results of this study show that oleic acid is as effective as linoleic acid in lowering LDL-C levels in normo triglyceridemic patients. Oleic acid seemingly reduces HDL–C levels less frequently than does linoleic acid. Neither type of unsaturated fat had striking effects on lipoprotein levels of hypertriglyceridemic patients.

Another group of workers who investigated the monounsaturated oil were Sortori et al (1986) who assessed the effects of low factor diets on lipids and platelets in 23 middle aged patients with high atherosclerosis risk for 8 weeks. The olive oil diet had a P:S ratio of 0.33 versus 1.28 for the corn oil diet. Plasma totalcholesterol was reduced with corn oil, but HDL-C levels were lower with corn oil and unchanged or raised by olive oil. Plasma apolipoprotein B levels were equally reduced by both diets, apolipoprotein AI and apo AI: B ratio rose only with olive oil. An olive oil diet, the authors concluded, with a moderate intake (about 30% of total calories) leads to favourable plasma lipoprotein and platelet changes.

Berry *et al* (1986) studied the relationship of dietary fat to plasma lipid levels as studied by factor analysis of adipose tissue fatty acid composition in a free living population of middle aged American men. When monounsaturates (animal fats) were increased, an increase in plasma TG, TC and VLDL–C was seen An increase in poly unsaturates (vegetable oils) was associated with lowered TG, VLDL–C and HDL–C but increased LDL-C. Based on contribution of factors to variance, the authors stated that adipose tissue composition, and by implication, the type of dietary fat intake, explains only a small proportion (1–19 per ecnt) of the variance in plasma lipids in normolipidemic subjects.

However, the lipid research clinics coronary primary prevention trial illustrated (Glueck *et al*, 1986) that the total and low density lipoprotein cholesterol in hypercholesterolemic men

could be very significantly lowered with dietary modification. The sample consisted of 3806 men and in addition to diet, othe correlates studied were weight, smoking, drugs etc. Decrease in SFA and cholesterol and increases in PUSFA were consistently associated with reduction in total cholesterol and in LDL–C. With P/S ratio of 0.8 and a daily cholesterol intake of 400 mg. in 6494 men who were instructed on this diet during screening, the mean total cholesterol decreased from 283 to 267 mg/dl and the mean LDL–C levels decreased from 207 to 190 mg/dl after one month. Weight loss, reduction in dietary intake of SFA and cholesterol and increase in the dietary intake of polyunsaturated fat were significant independent predictors of the plasma cholesterol reduction. Decrements in SFA were significantly associated with decrements in total C and LDL–C. This was maintained throughout the 7 years trial period. Increments in PUSFA were associated with decrements in total–C and LDL–C, but these relationships were generally weaker than those for SFA (Glueck *et al*, 1986). The quality of fat ingested was a far stronger determinant of SC levels (Ahrens, 1985; Williams *et al*, 1986, Suk Y Oh and Monaco (1985) showed in their study that regardless of dietary cholesterol levels, the plasma cholesterol levels were significantly decreased by the diets high in P/s ratio. Mc Namara et al (1987) examined the effects of dietary fat and cholesterol homeostasis in man. In 69 per cent of their studies the subjects compensated for the increased cholesterol intake by decreasing cholesterol fraction absorption and/or endogenous cholesterol synthesis. The authors stated that plasma cholesterol levels were more sensitive to dietary fat quality than to cholesterol quality.

The cholesterol lowering capacity of polyunsaturated fatty acids has been experimented upon by many investigators. Beynen and Katan (1985) explained that the PUSFA in the diet may lower serum VLDL concentrations, because the liver preferentially converts PUSFA into ketone bodies instead of into VLDL tryglycerides. The formation of VLDL apo B is less with linoleate rich than with more SFA diets.

It has been shown that dietary fat down regulates LDL-receptor activity, thereby reducing the uptake of LDL from the plasma into the cell and so increasing plasma LDL concentration (Rifkind, 1986).

Significant positive correlations with CHD were shown for the dietary lipid score (based on percapita consumption of cholesterol, SFA&PUSFA) and sugar. The risk of dying from CHD was significantly related to the lipid score (sum of dietary cholesterol plus SFA minus PUFA). Consumption of plant foods reflecting PUSFA and fiber was inversely correlated with CHD mortality. By modifying the quality and quantity of fats and restricting dietary cholesterol, certain beneficial effects in the lipoprotein fractions have been shown. Based on a few studies the following conclusions can be drawn. 1. LDL-C levels are lowered substantially by marked reduction in fat intake which is largely independent of P : S ratio. 2. Quantitatively similar lowering of LDL is achieved by a more modest restriction of fat intake to about 30 per cent energy provided the P : S ratio is raised to at least 1, 3. HDL-C may also fall by almost as much as the LDL-C by increasing PUFA intake. 4. A low fat diet (20% energy or less) usually lowers HDL-C by as much as LDL-C.

It is important to recognise that most studies of low fat diets have shown a rise in VLDL TG, which may compound the potentially undesired effect of lowering HDL-C. Such rises in the TG concentration have tended to occur when fat intake has been restricted to <25% energy. At fat intakes of 30 - 40% energy, VLDL levels are infact generally lowered by diets rich in PUFA, although sometimes such changes are not great (Nestel, 1987).

Linolenic acid may be converted to eicosapentaenoic acid (C 20 ; 5 w-3) in mammals. The hypotriglyceridimic and hypocholesterolemic effect in human beings of dietary fish oils containing w-3 fatty acids appears to be due to inhibition of VLDL synthesis (Harris *et al*, 1983). Mortality from coronary heart disease was more than 50 per cent lower among Netherlands men who consumed at least 30g fish per day than among those who did not eat fish (Kromhout *et al*, 1985).

The n-3 dietary fatty acids; αLinolenic acid, Eicosa Pentaenoic acid (EPA, 20 : 5) and Docosa Hexanic Acid (DHA, 22 : 6, n-3) are capable of lowering Lipoprotein cholesterol even when the intake of cholesterol is high (Nestel, 1986; Nestel and Fracp, 1986). The biochemical and metabolic changes observed after n-3 fatty acid feeding are generally consistent with reduced development of cardio vascular disease (Herold and Kinsella, 1986.)

The n-3 fatty acids give rise to different families of eicosanoids such as prostanoids and leucotrienes which are regulators and modulators of many biological systems in man. Dietary EPA gives rise to the formation of the three series of prostanoids (PGI_3 and TXA_3) in man. This leads to a marked change in the two or three series of prostanoids with major consequences for at least thrombogenic mechanisms. Swansonsor *et al* (1988) stated that n-3 fatty acid consumption resulted in the incorporation of eicosapentaenoate and docosa hexa enoate in tissue lipids which altered thromboxane A_2 (TXA_2) and prostacyclin (I2) and this factor is important in ameliorating coronary Artery Disease.

At high intakes (2-20 g EPA daily), an increase in bleeding time of whole blood, a blood pressure lowering effect and a decrease in the aggregability of platelets has been reported. An anti inflammatory effect mediated through the inhibition of leukotriene B_4 production in leucocytes also required at least 3g EPA daily. The n-3 fatty acids have profound influence on the production of both thrombogenic and antithrombogenic compounds of blood platelets and the endothelium of the artery. Daily consumption of 10-15 g of fish oil extract representing 3-5g n-3 fatty acids is probaly adequate to control moderate hypertriglyceridemia and in modifying the raising effect of dietary cholesterol, (Schacky *et al*, 1985 and Nestel, 1987).

1.3.3 Dietary Cholesterol and Serum Cholesterol:

Since the early years of this century, attention was directed towards the high blood cholesterol levels in populations habitually consuming high cholesterol, high fat diets. Significant correlations were shown between the high fat and cholesterol content of diet and incidence of CHD.

Connor *et al* (1961) stated that dietary cholesterol was effective in regulating blood cholesterol levels. According to Keys, exogenous cholesterol increases the serum levels in proportion to the square root of its concentration in the diet. The daily intake of cholesterol in an average diet (Western) may vary from 200 to 800 mg, while the endogenous synthesis can supply daily approximately 2000 mg or even more. Endogenous synthesis of cholesterol can be inhibited by the excessive intake of dietetic cholesterol through a feedback mechanism (Angelico, 1975).

For cholesterol intakes between 50 and 1550 mg per day SC concentration is a function of the square root of the dietary cholesterol (Grande *et al*, 1965). The effect of changing the cholesterol content of the diet can be illustrated by the following example. Assuming a cholesterol intake of 250 mg/1000 calories, a reduction of 30 per cent in the cholesterol intake (to 175 mg/1000 cal) will produce a decrease of SC concentration of only 4 mg/100 ml (Grande, 1966).

With increased P/S ratio and decreased cholesterol in the prudent diet, the anti-coronary club, New York showed incidence of new coronary events in their experimental group (Roine, 1972).

The investigations of wells and Bronte-Stewart, (1963), Keys and Coworkers, (1965), and Hedgsted *et al*, (1965), have shown that dietary cholesterol intake, if it exceeds a certain level, can cause an increase in serum cholesterol.

According to the recommendation of American Heart Association, cholesterol intake should be reduced to 300 mg/day in persons with high cholesterol levels (Muller, 1973). Such a dietetic measure seems to be rather important in countries, in which the dietary cholesterol intake is generally high eg., in USA where it is around 650 mg/day. It can be calculated by the formula described by Keys aud coworkers (Grade *et al*, 1965), that 100 mg dietary cholesterol causes an increase of about 6 mg per cent of serum cholesterol. This means, that for example a regular daily intake of one egg containing about 300 mg cholesterol raises the serum cholestesterol by 18 mg per cent, i. e. nearly 10 per cent of the so called normal serum cholesterol value.

In some studies, no relationship between dietary cholesterol and serum cholesterol levels was found (Dawber *et al*, 1982; Buzzard *et al*, 1982; Liebman and Bazzarre, 1983). Buzzard *et al* (1982) stated that, some presumably healthy responders may show sensitivity to high consumption of eggs by showing increased plasma cholesterol levels. On the other hand there are non-responders who show little or no increase in SC in response to high egg ingestion. The difference between responders and non-responders may be related to control of the production of hydroxymethyl glutanyl coenzyme A reductase, the enzyme that regulates cholesterol biosynthesis. The differences in the capacity of these compensatory mechanisms – suppression of synthesis and increased fecal steroid excretion–that try to prevent accumulation of cholesterol in the body at high intakes are evidently involved.

Plasma cholesterol becomes attenuated on diets with high P:S ratio or low fat content (Pyorala, 1987). Liebman and Bazzarre (1983) studied the effects of egg consumption in vegetarian and non-vegetarian males, on plasma lipids. Inspite of widely differing egg consumption, no relationship was observed between egg cholesterol intake and plasma lipid levels. Instead, total fat exerted an important influence upon the plasma lipid levels i.e. total cholesterol and triglycerides were 11 and 21 per cent lower and the mean HDL–C levels 14 per cent higher in low fat vegetarians (23 to 33% Kcal from fat) compared to high fat vegetarians (35 to 48%). The high fat intake in vegetarians was partially due to a high reliance on dairy products. Diets characterized by high intakes of dairy products are not always associated with markedly lower plasma lipid levels.

Three factors influence the effect of dietary cholesterol on serum cholesterol in humans, 1) composition of the diet with respect to other nutrients, including PUSFA and unknown anti or pro – hypercholesterolemic agents (2) the base line level of dietary cholesterol from which observations are made and (3) individual variability. Some people are hyper responders and some are hypo responders. This responsiveness relates, in general, to the extent to which exogenous cholesterol interacts

with the cholesterol homeostatic system in each individual. Cholesterol biosynthesis is more tightly regulated in some people than in others.

Feeding 3-6 eggs per day increases plasma LDL and HDL cholesterol levels in about 50 per cent of normo–cholesteremic subjects. This effect is blunted by increasing the dietary P/S ratio to 1.0 (Nut· Rev., 1985). Applebaum – Bowden *et al* (1984) demonstrated that high levels of dietary cholesterol i. e. 1034 mg/day, can down regulate the LDL receptor in humans which contributed to an increase ($P<0.02$) in LDL cholesterol level.

Suk Y oh and Miller (1985) demonstrated a wide variability of plasma cholesterol levels and lipoprotein cholesterol with dietary egg ingestion. Based on response, the subjects were divided into hypo and hyper responders. The amount of cholesterol in association with HDL_2 and HDL_3 (subclasses of HDL) in the hyper responders was greater than the hyporesponders. Hyper responders had a lower initial plasma cholesterol level. Accordingly the hyper responders in this study were considered as at lower risk for developing premature CHD due to cholesterol consumption.

In his critical analysis of serum cholesterol response to dietary cholesterol, Keys (1984) stated that dietary cholesterol affects serum cholesterol level and some experiments which reported no relationships, were not critically designed, taking the substantial effect of quality of fat into consideration. If the diet is fairly high in cholesterol, (eg : 300 mg/1000 kcal), the effect of cutting the dietary intake of cholesterol in half, with no other change, can be expected to bring only a reduction of about 7.6 mg/dl of cholesterol in the serum. Keys (1984) mentions that changes in fatty acid composition of the diet are more consequential. If saturated fatty acids provide 18 per cent of the calories in the diet, then by reducing that amount to half, with isocaloric replacement by simple carbohydrate, one can expect to produce an average reduction of some 23 mg/d l in the SC. Add the effect of cutting in half the dietary cholesterol and the total effect should be, on an average, more than 30 mg/dl decrease in SC, which is certainly not negligible.

Re–evaluating the serum cholesterol response to dietary cholesterol, Hegsted (1986) stated that over the range of cholesterol intakes of practical interest, O – 400 mg/1000 kcal – the usual response is approximately linear, each 1 mg/1000 resulting in an expected increase of serum cholesterol of 0.1 mg/dl. With a 2500 kcal diet, an increase in intake of 100 mg/day would be expected to increase SC by 4 mg/dl. While applauding Hegsted's new equation Keys (1986) comments stating that their old square root equation could be still preferred for accuracy and ease of calculation.

1.3.4 Dietary Carbohydrate and Serum Cholesterol and Serum Triglyceride Levels.

High dietary carbohydrate intake is accompanied by high fasting levels of circulating endogenous triglycerides (TG). Another influence upon serum triglyceride is the type of dietary carbohydrate; dietary sucrose provokes higher endogenous TG concentration, than does either dietary starch or dietary glucose taken in similar amounts. This effect appears to be due to the fact that sucrose yields equal amounts of glucose and fructose. Fructose is converted more readily into endogenous TG than glucose. considerable amount of evidence for the involvement of sucrose in the causation of CHD has accumulated over the past years.

A statement of the British Nutrition Foundation referred to sucrose in moderate amounts as favourable to CHD. A couple of centuries ago, the average consumption of sugar in England and America was of the order of 10g/day. But the consumption in America increased to about 125 g/day. Such increased consumption of sugar over a period of time can produce changes including an increase in SC and STG (Nutrition Today, 1970). High sucrose feeding increased plasma TG levels, produced hyper insulinism and deteriorated glucose tolerance. High platelet aggregation and high lipid in the aortic wall of rat was also seen (Yudkin, 1972 and Roberts, 1973).

When most normal men increase the proportion of carbohydrates in their diet, the level of TG in the fasting serum

increases at first before gradually returning to normal. In those whose fasting serum TG level is raised, an increase in the proportion of dietary carbohydrate causes a more marked rise which will not, if the increased carbohydrate intake persists, return to normal values. This group of patients are called carbohydrate – induced hyperlipoproteinimic of Type IV. As this carbohydrate induced hypertriglyceredemia is associated with vascular disease, it is obviously necessary to advise such a person to reduce the intake of carbohydrate (Mac Donald, 1975).

In man, when excess calories are in the form of carbohydrates, glucose is first converted into TG in the liver and secreted into the circulation as VLDL. A very low fat, high carbohydrate diet causes an approximate doubling of triglycerides (Albrink, 1973). A very high carbohydrate diet (75% of total calories) causes an almost universal increase in TG concentration, of uncertain duration, perhaps a permanent elevation in susceptible individuals. Increased hepatic synthesis of TG from carbohydrate is the probable cause. Two factors are accounted for basal hypertriglyceredemia, i.e., over production and impaired removal of TG, the removal defect merely being exaggerated by high carbohydrate diet (Albrink, 1973).

Studying the low fat versus low carbohydrate diet in the treatment of type IV hyperlipoproteinemia, Sommariva *et al*, (1978) showed that reduction of carbohydrate intake is followed by a significant decrease in serum triglycerides and pre B–lipoproteins. A low fat produced a significant fall in serum cholesterol and B–lipoproteins, in addition to triglycerides. Thus reductions in serum lipid levels observed with a low fat and a low carbohydrate diet were significant.

The effect of changes in the amount of carbohydrate (45 or 65 per cent of total energy) and in the source of carbohydrate (sucrose or corn syrup) on plasma triglyceride and cholesterol concentrations in eight healthy males was studied (Hayford *et al*, 1979). The fasting plasma TG concentration increased significantly during ingestion of the high carbohydrate diet ($P<0.005$), but was not significantly influenced by the source of carbohydrate calories. The 45 per cent carbohydrate diets induced larger meal associated plasma TG variation than the 65 per cent diets. With the 60 per cent carbohydrate diet. Lin et al (1983) reported that the TG levels were never lower.

But the kind of carbohydrate that is given is important, as Lewis *et al* (1981), tripling the amount of dietary fiber, reversed the increase in VLDL–TG and decrease in HDL_2 – cholesterol produced when an equally high carbohydrate diet (50 per cent of total calories) containing more conventional foods was used. When high carbohydrate diets are to be prescribed, the sucrose component is also to be considered in such diets.

1.3.5 Dietary Protein and Serum Cholesterol and Serum Triglyceride Levels

In protein deficiency, the plasma cholesterol is low, but when protein and amino acid requirements are met, no convincing change in the plasma cholesterol level with difference of quantity or source of protein intake was shown. The cholesterol lowering effect of high protein diet was attributed, in part, to sulphur containing aminoacids which the protein provides (Fillios and Mann, 1954).

In general, protein derived from animals have greater tendency to elevate plasma cholesterol than proteins derived from plants, which is partially attributed to the amino acid composition. This follows the trend that in human populations atherosclerosis and heart disease are more prevalent in countries where more animal protein is eaten. Controlled experiments with human subjects are providing evidence that replacing animal protein in the diet by plant protein can reduce the level of plasma cholesterol (Devadas, 1979; Sirtori *et al*, 1975).

Uma Benerjee and Chakrabarti (1973) reported an animal experiment, in which pulse protein at 12 per cent level showed more cholesterol in liver and blood compared to pulse protein at 18 per cent level. But when these were supplemented with a mixture of methionine, lysine, threonine and tryptophan each at 0.3 per cent level, the animals showed a lowered cholesterol content in different tissues examined.

The hypocholesterolemic effect of pulses was investigated by Soni *et al* (1982). Among four different pulses, bengal gram has been shown to be more potent hypocholesterolemic agent followed by blackgram and greengram. When pulses were used as dietary protein sources, the rate of catabolism of cholesterol was higher (assessed by fecal bile acid) as compared to the control group.

Usha chandrasekhar *et al* (1983) investigated the effect of bengal gram further, by subjecting it to processing, to study its effect in lowering serum cholesterol and triglyceride levels and histopathological alterations of heart, aorta and liver. There was significant decrease in the cholesterol and triglyceride levels between the atherogenic and experimental phases. There was also a significant difference ($P < 0.01$) between roasted and raw bengal gram. The animals on roasted bengalgram diet showed reduced levels of cholesterol than those on raw bengal gram diet. The roasted bengalgram or processed pulses have a beneficial effect in lowering cholesterol and triglyceride levels. Histo pathological changes of the heart, aorta and liver were observed. They indicated accumulation of fat cells in coronary artery, thickening, vacuolation and lipid accumulation in aorta and fatty degeneration and absence of glycogen vacuolation in liver. With a raw bengalgram diet, histopathology of specific tissues indicated an accumulation of fat cells in the coronary artery, inflammation of myocardium and mild but definite fatty degeneration of the liver. These were not seen in rats fed roasted bengalgram diets thus bringing out the superiority of pulses, especially roasted legumes in maintaining normal tissue histopathology.

1.3.6 Ascorbic Acid and Serum Cholesterol Levels

Ascorbic acid is believed to play a part in cholesterol metabolism. High ascorbic acid protects against atherosclerosis. Deficiency of ascorbic acid is a contributing factor in the development of myocardial, aortic and cerebral atherosclerosis (Shaffer. 1970).

Vitamin C is concerned with the maintenance of normal vascular function and a deficiency of it may cause vascular disease. There is a general agreement that ascorbic acid has a major role in the integrity of the ground substance of the arterial intima. A deficiency of the vitamin results in the disturbance of the ground substances which is effective in producing lesions in animal arteries which are morphologically identical to human atherosclerosis. Correction of this deficiency prevents and generally decreases the lesion of experimental atherosclerosis, suggesting that ascorbic acid metabolism is related to the pathogenesis of atherosclerosis (Krumdick and Butterworth, 1974).

In subjects whose ascorbic acid intake is low, but not necessarily low enough to cause other manifestations of deficiency, serum cholesterol may rise. Possibly a marked seasonal variation of serum cholesterol observed in some rural populations is partly due to a marginal intake of ascorbic acid (Karvonen, 1972). Pelletien and Keith (1974) stated that the bioavailability of ascorbic acid from synthetic L-ascorbic acid was slightly superior to that from orange juice. The ascorbic acid in serum leucocytes and urine was measured. A slightly higher urinary excretion of the vitamin after orange juice administration explains in part the lowering of the serum levels.

Ginter (1974) stated that care should be taken to prevent a state of latent ascorbic acid deficiency and thereby forestall any disorder in the process of cholesterol transformation to bile acids. He showed that such a disorder in experimental animals leads to hypercholesterolemia and atherosclerosis and this may possibly apply also to the humans. The fact that in countries with high living standards the intake of vitamin C in certain groups of the population is very low, only goes to underline the urgency of this problem.

Long term administration of ascorbic acid prevented the progression of pathology of atherosclerosis in rabbits (Sokoloff *et al*, 1966). Some hypercholesterolemic patients showed decrease in their cholesterol values with a high dose of ascorbic acid

supplement (Samuel and Shalchi, 1964). Spittle (1972) stated that vitamin C decreases the cholesterol level in normal people too.

1.3.7 Dietary Fibre and Serum Lipids

Dietary fibre is a ubiquitous component of plant foods and includes material of diverse chemical and morphological structure, which is resistant to digestion by the secretions of human gastrointestinal tract.

Dietary fiber is defined as the sum of lignin and the non starch polysaccharides. Dietary fiber includes both water soluble and water insoluble substances and many of these are non-fibrous in the accepted sense of the word (Vahouny and Kritchevsky, 1982).

Ingestion of certain fibres, especially the water soluble fibre is accompanied by a significant reduction in SC concentration. Increased fecal loss of bile acids and cholesterol may be the reason to some extent (Anderson, 1978 ; Keys *et al*, 1978).

The intestinal mucosa synthesizes chylomicrons, very low density lipoproteins and high density lipoprotein components. Fibre ingestion may alter the synthesis and secretion rates of these lipoproteins. A diet rich in fibre containing foods is by nature high in complex carbohydrates and vegetable derived protein. By displacement of food items that contribute saturated fat and cholesterol, fiber may indirectly result in lower plasma lipid levels. Intake of dietary fibre and percentage of calories consumed as fat are inversely related in a normal population (Kay and Truswell, 1977). Many types of isolated and native fibres have been shown to bind bile acids and cholesterol in vitro. Absorption of bile acids is influenced, by the physical and chemical form of the fibre, the polarity of the bile acid. and the type of bile acid micelle and the osmolarity of the intestinal contents (Spiller *et al*, 1980).

Saponins are widely distributed in the plant kingdom, especially in legumes. Saponins are powerful surfactants in

which a disaccharide and a steroid of triterpene are combined in one molecule. Dietary saponins remain within the Gastro Intestinal tract and are not absorbed into the blood stream. Thus, they could induce association between fibre and bile salts particularly, as they are known to interact strongly with bile salts and with sterols in general. The dietary saponins may lower the SC levels and consequently the risk of heart disease (Topping *et al*, 1977). The increase in the incidence of heart disease in Western societies seems to coincide with a decline in the consumption of saponin rich legumes.

In a study of 200 healthy men, Keys *et al* (1980) reported that men in the lower tertile of the plasma cholesterol and TG distribution were consuming significantly more dietary fiber and significantly fewer calories as fat.

The effect of dietary fiber on plasma cholesterol concentration is generally believed to be largely mediated by enhanced fecal excretion of bile acids. Pectin, guar, cellulose and various food derived fibres significantly increased fecal bile acid output under diverse conditions. Mucilageous fibres form gels in the small intestine that could interfere with the absorption of both cholesterol and bile acids (Kretch *et al*, 1979). The colonic production and absorption of short chain fatty acid metabolites of fibre may alter hepatic fatty acid and TG metabolism (Anderson *et al*, 1978).

The effect of various types of fibre on bile acid excretion in man finds an average increase in fecal bile acid excretion of 60 per cent (Kritchevsky, 1982). Fiber may exert its hypocholesterolemic properties by other mechanisms including intestinal transit time, lipoprotein formation, transport and metabolism and sites of absorption.

In an investigation on healthy normolipidemic men, fiber components pectin, cellulose and lignin did not significantly change the serum lipids i.e., total cholesterol, TG and HDL in 4 weeks. The authors Hillman *et al* (1985) suggested that lignin contributed to a fall in higher serum cholesterol levels, and therefore it may be worthy of study in hyperlipidemic subjects, for a longer term and with fibre from different sources.

Moore et al (1988) studied the beneficial short term effects of unprocessed wheat bran on lipid metabolism in man. 0.15 g of unprocessed wheat bran/kg body wt/day, for 6 weeks was given to healthy, 18-22 year old volunteers. After 6 weeks, HDL-C was increased by 46 per cent and LDL-C decreased by 25%. However. fasting concentrations of plasma total-C and TG were unchanged. Vighe et al (1987) studied the effects of pectin, bran and cellulose on serum lipids and lipoproteins in rats fed on a low or high fat diet. Rats were fed for 6 weeks a diet with low fat content (50 g/kg) and a diet rich in fat (250 g/kg). In both cases the basal diets were either fibre depleted or supplemented with cellulose, wheat bran or low methoxyl pectin. When low fat diet was given, the low methoxyl pectin displayed the most hypo cholesterolemic effect and decreased the cholesterol content of the VDL and LDL. This may be due to binding of bile salts, phospholipids and cholesterol; due to induced changes in the viscosity of intestinal contents, a property directly linked to the molecular properties of the different pectins and due to the fact that pectins might alter the ultrastructure and some functions of the intestinal mucosa. These combined influences might impair or delay the intestinal absorption of cholesterol and fats, decrease intestinal lipid synthesis and lower the lymph lipid out put which inturn might decrease liver cholesterol and TG accumulation and promote beneficial changes in serum lipid and lipoprotein fractions. But a high fat diet would tend to inhibit these effects. Wheat bran and cellulose exerted no hypocholesterolemic effect. The magnitude of the effect of each individual type of fiber is dependent on the fat and cholesterol content of the diet suggesting the existence of different mechanisms of action.

1.3.8 Miscellaneous Items

Coffee : A significant positive correlation between coffee drinking and serum lipid concentration was found by Little *et al* (1966), while tea drinking tended to have a negative correlation. Caffeine in coffee elevates the serum lipids. However, in an Indian study (Srimathi *et al*, 1981), drinking 4 to 5 cups of coffee with milk and sugar did not elevate serum cholesterol and triglycerides either in rats or in human volunteers (n = 20). But

the studies reported from the west do show a good relationship between the coffee consumption. especially between black coffee and high cholesterol levels.

Thelle *et al* (1984) divided 17 healthy volunteers into 2 groups. The experiment lasted 9 weeks with coffee period and no coffee period. The cholesterol level decreased from 206 to 190 mg per dl during the no coffee period and increased there after to 235 mg/dl in the coffee period. A daily intake of 6 or more cups of boiled coffee increases the serum cholesterol level in healthy subjects. Thelle et al (1983) examined the relation between coffee consumption and serum cholesterol, triglycerides and HDL-C in 7368 men and 7213 women. After a covariance analysis, the coffee-cholesterol relation remained strong and statistically significant ($P<0.0001$) after adjustment for other important risk factors. The authors concluded that coffee consumption is a major contributor to the variation in levels of total cholesterol,

Forde *et al* (1985) observed a similar trend in their experiments on 33 men. The authors concluded that abstention from heavy coffee drinking is an efficient way of reducing serum cholesterol concentrations in men with hypercholesterolemia.

The effect of caffein intake on blood pressure seems to be controversial. Caffein is believed to bring about a change in the renin, angiotensin, aldosterone and catecholamine levels in the body. Higher intakes of caffein increases the renin concentration and is antinatriuretic (Medeiros, 1982). Robertson *et al* (1978) found that a single dose of 250 mg of caffein could increase the blood pressure of subjects by 14/10 mm Hg.

In India too, studies to understand the effect of coffee, especially when it is drunk with milk and sugar and with lot of variation in the amount or in dilution, need to be carried out to relate it to serum cholesterol and thus to CHD.

Onion, Garlic & Ginger : Ingestion of onion and garlic results in hypocholesterolemia and increased fibrinolytic activity

of blood in alimentary lipemia (Bordia *et al*,, 1974; Jain, 1975). Bordia *et al* (1974) investigated the effects of the essential oils of onion and garlic in cholesterol fed rabbits and compared with the effects of clofibrate. The marked rise in serum cholesterol was significantly reduced by the essential oils of both onion and garlic. Garlic was even more effective than onion. The mechanism of the hypolipimic action of garlic is of increased excretion of the bile acids and sterols in the feces. Diminished tissue lipids suggest that it may act both by increasing the excretion of cholesterol end products and by affecting their endogenous synthesis in the liver. Recently, the usefulness of garlic as a hypocholesterolemic agent has been questioned. Sogani and Katoch (1981) reported that heavy garlic and onion consumption by heart patients resulted in increased serum cholesterol levels. Gupta et al (1987) demonstrated that 30 days of garlic supplementation increased the serum cholesterol levels in rabbits and withdrawal of garlic restored it to normal or pre experimental levels. Since the hypocholesterolemic effect of onion and garlic is still controversial, no definite conclusions can be drawn.

Janabai Giri (1984) assessed the effect of ginger on SC levels of rats fed cholesterol for 24 days, the added ginger restrained the increase of blood cholesterol level significantly. When ginger was administered for another 24 days, it decreased the serum and hepatic cholesterol significantly. It is evident from the results that ginger is definitely hypocholesterolemic, but needs to be taken daily for several days to obtain significant results. It has no immediate effect on serum cholesterol.

The foregoing studies on relationships and associations of various dietary constituents and serum lipid levels could be summarised as follows:

1. The total calorie level influences the body weight. Body weight is positivcly and strongly associated with higher serum cholesterol and triglyceride levels. This makes it necessary to recommend normal or ideal body weight.

2. There is close agreement among workers from various parts of the world, that a decrease in saturated fat, dietary cholesterol and an increase in polyunsaturated fatty acids and thus an enhanced P/S ratio can contribute to a significant fall in serum cholesterol and triglyceride levels. Though there is a controversy about dietary cholesterol. inview of variability in response between individuals, it is safer to restrict the dietary cholesterol to about 300 mg per day.

3. Dietary carbohydrate has been shown to be related to serum triglyceride to a greater extent than to serum cholesterol. The mono and disaccharides, especially fructose and suc.ose, exert a more harmful effect. Therefore, they are to be restricted in the diet. The carbohydrate content could be around 60 per cent of the total calories and preferably given with fiber present (complex carbohydrate).

4. The protein content could be obtained from pulses also, in addition to animal protein and dairy sources. Pulses supply considerable amount of proteins and they have been shown to possess hypocholesterolemic property. In Indian diets, a major proportion of protein requirement is met from pulses.

5. Fresh fruits and vegetables supply good amounts of ascorbic acid which is shown to prevent the process of atherosclerosis. In addition, fruits and vegetables supply high fibre content. Vitamin C and fibre are inversely associated with hyperlipidemia.

6. Various flavoring agents like onion, garlic and ginger, in the long run may bestow certain beneficial effects which should be appreciated.

Nissinen and Stanley (1989) quoted Connor and Connor who examined the association between the intake of several nutrients in the diet and the mortality rates from Coronary Heart Disease for 30 different countries. The results show that dietary cholesterol, animal protein, animal and total fat, meat

and eggs as well as total calories and sugar correlate positively with the mortality among men aged 55–89 years. Starch and vegetable protein correlate negatively whereas plant sterols, fish, vegetable fat and vegetables have no correlation.

Pietinen et al's (1989) North Karelia (Finland) project is the first community based intervention program aimed at controlling cardio vascular diseases. The changes in diet and serum cholesterol levels were more pronounced in North Karelia than in the reference area during the first 5 year period. The project put pressure on the food industry to provide for example low fat dairy products and to decrease the salt content of breads, sausages and catered food. This contributed to significant reduction in serum cholesterol levels, However changing lifestyles in the population are a slow and not always a continuous process. Now ideas for intervention techniques are constantly needed. Promotion of good nutrition and healthier lifestyles in general calls for continuous efforts and good co-operation between researchers, health educators, legislators and the food industry to make changes that are feasible for people.

An individual eats twice, thrice or four times a day. depending on his/her dietary pattern or life style. The cumulative effect and the synergistic action of various components of the diet affect the individual to a remarkable extent. With this point in mind, appropriate, suitable, adaptable and acceptable manipulation of the eating style is to be resorted to, if he/she wants to be spared of the dreaded morbidity or mortality due to IHD.

MATERIALS AND METHODS

The present study was carried out with the following objectives:

1. to estimate Serum Cholesterol (SC) and Serum Triglyceride (STG) levels in a sample of the student, the teaching and the non teaching population of Sri Venkateswara University, belonging to the 20–29, the 30–39 and the 40–54 year age groups;

2. to assess the mean nutrient composition of the food intake of these subjects;

3. to examine the relationships of each one of dietary constituents to SC and STG levels;

4. to assess the association of each of the risk factors namely age, obesity, hypertension, cigarette smoking, family history and coronary-prone behaviour to SC and STG levels; and

5. to study the combined contribution of the aforesaid variables to SC and STG levels.

The realisation of these objectives required that

a) an appropriate sample of subjects be drawn;

b) the serum cholesterol and serum triglyceride levels of these subjects be measured;

c) the food intake of the subjects be recorded;

d) the obesity index of the subjects be calculated, taking the height and weight of the subject into consideration;

e) the blood pressure, systolic and diastolic be measured.

f) the coronary prone behaviour of the individuals be assessed; and

g) the number of cigarettes smoked, the family and medical history and the other bio-data be recorded.

2.1 The Sample

Three hundred men, drawn through a purposive sampling procedure, constituted the subjects of the study. Of the three hundred, one hundred belonged to the age group 20–29; one hundred belonged to the 30–39 age group, while the third hundred were between 40 and 55 years of age. Post-graduate students and Research scholars constituted the sample in the 20–29 age group. Individuals in the 30–39 age group belonged mostly to the teaching faculty. The third age group (40–55) consisted largely of the teaching and non-teaching staff. All the subjects of the sample belonged to Sri Venkateswara University. These subjects were picked from the staff and students lists such that the sample in each age group included individuals with positive family history, smokers, as well as those who were neither smokers nor had positive family history (normal). Care was taken to exclude individuals with diabetes mellitus, kidney, liver and heart diseases. The information on these aspects provided by the subject to oral questions formed the basis for exclusion from the sample.

2.2 Collection of Blood Samples and Other Relevant Information

First, the subjects were contacted in order to establish rapport. The objectives of the study were explained to them and they were requested to extend their full and whole-hearted co-operation. Since blood samples were needed for bio-chemical analysis, certain practical problems in convincing the subjects were encountered. Though most of the subjects accepted to give samples of blood, a few subjects were reluctant to do so, despite the fact that they were informed of the usefulness of getting their cholesterol values assessed. Ultimately, some yielded after

persuasion, but a few did not agree to the idea of giving blood samples! The desired number of subjects, however, was maintained.

Four days prior to the collection of blood samples, the subjects were given the diet questionnaire and a stainless steel cup. They were requested to record for each meal the quantity of intake of various food items (recipe form) in terms of level cups in the appropriate columns of the questionnaire daily, for three consecutive days and report at the Sri Venkateswara University Health Centre on the fourth day at 7 A.M. to give blood samples. Fasting blood samples were drawn into sterile syringes with the technical assistance of the health centre staff. About 5-8 ml of blood was drawn, transferred into labelled, clean glass vials and allowed to clot for about 2 3 hours. Precautions were taken to prevent hemolysis (Lamberg and Rothstein, 1978).

Soon after they gave the blood samples, the subjects were given a cup of coffee. The height and weight were recorded. Then the other questionnaires were administered. Information was obtained on smoking behaviour, family history, clinical history, coronary prone behaviour and other aspects. The entire session took about an hour. A couple of days later, the subjects were informed of their cholesterol and triglyceride values. Dietary advice was given to such of those whose values were on the high side.

2.3 Preparation of Serum for Cholesterol and Triglyceride Assay

The collected blood samples were allowed to clot. After 2–3 hours, when the serum had separated, it was transferred to centrifuge tubes and centrifuged for 20 minutes at 300 rpm. The supernatant layer of serum was taken into clean dry labelled tubes. The appearance of the serum and turbidity, if any, was noted. The serum was stored under refrigeration for the subsequent estimation of cholesterol and triglyceride. These estimations were carried out within 24–48 hours after separating the serum.

2.4 Estimation of Serum Cholesterol Levels

Total serum cholesterol was estimated by the Carr and Drekter (1956) method, in which glacial acetic acid does not interfere with the "Liebermann - Burchard" reaction. Acetic anhydride fulfils the multiple functions of cholesterol extraction, precipitation of serum proteins and furnishing a medium suitable for colour development. This also facilitates production of the necessary anhydrous system before addition of colour reagent, concentrated sulphuric acid and glacial acetic acid reagent which provides adequate sensitivity and stability of colour for the conditions of analysis. Generally duplicate samples of serum were drawn in the determination of cholesterol levels. If necessary, a triplicate sample was drawn.

2.4.1 Principle

Cholesterol in the serum is extracted into acetic anhydride in the presence of acetic acid. On treatment with a modified "Liebermann–Burchard," reagent, it develops a green colour, the intensity of which is proportional to the concentration of the substance. This is compared with a standard. The colour was read in Spectronic–20 (Bausch and Lomb) at wave length 630 nm. The standard curve was plotted with concentrations ranging from 0.1 to 0.8 mg cholesterol.

A recovery study was done to check the accuracy of the method. The recovery values obtained were 100 and 100.12 per cent.

2.5 Estimation of Serum Triglyceride Level

Serum triglyceride level was estimated by the method of Fletcher (1968) as modified by Foster and Dunn (1973).

Principle: The glycerides contained in an Isopropanol extracts of serum are hydrolysed by saponification. The free glycerol is then oxidised to HCHO. This reacts with acetyl acetone and ammonia to give a yellow compound, the absorbance of which is measured. Phospholipids are removed by absorption on alumina mixture prior to saponification.

Duplicate samples of serum and standard solution of triglyceride were used for estimation of triglyceride. If necessary, a triplicate sample was used.

A standard curve was plotted with concentrations of 300, 150, 75, 50 and 30 mg triolein per dl. The triolein standard used was from Sigma chemical company, U.S.A.

A recovery study was done to check the accuracy of the method. The recovery of triglyceride was 99.6 and 100.9 per cent.

2.6 Dietary Data

2.6.1 Collection of Dietary Data

Data pertaining to dietary intake was collected using a questionnaire. It consisted of a number of questions to elicit information about the diets consumed. The draft questionnaire was tried on a sample of subjects belonging to the three age groups and their comments obtained. It was scrutinised for adequacy by two nutritionists and the Research Supervisor who were famitiar with diet surveys. The diet questionnaire is given in appendix.

The food consumed by the subjects was recorded in the diet questionnaire. For this purpose, all the subjects were instructed and requested to measure the cooked foods (recipe form) they consumed at each meal in terms of a stainless steel cup (commonly used in food service) that was provided to them. They were instructed to take level measures of the food and indicate in the questionnaire, the number of cups taken. For items like idli, puri and dosa, they were asked to indicate the number consumed and the size. They were asked to record the food (recipe) intake for every meal (breakfast, lunch, dinner etc.) in the blank columns of the questionnaire meant for the purpose. They wree further requested to name the recipe and its quantity consumed at each meal for the three consecutive days preceding the day on which the blood sample was drawn. Also, they were asked to record the quantity of visible fat/oil intake each day in terms of the steel cup or a standard tea spoon.

2.6.2 Standardisation and the Calculation of Nutrient Composition

The stainless steel cup that was used in the study was standardised in the laboratory. The common recipes that were consumed by all the subjects in the sample were rice, sambar, dhal, rasam, curd and vegetable curries. Generally these recipes are prepared in the homes using a standard method and ingredients. Adopting the same method, these recipes were prepared in the laboratory. The raw weights of ingredients used, the cooked weights of the recipes and the yield in terms of the steel cup were recorded. For breakfast recipes like idli, dosa, poori etc,, the raw weight equivalents for the number and size of the recipe were determined.

From the quantities of raw weights of ingredients recorded, the nutrients like carbohydrate, protein, fat (invisible), vitamin C, fiber and the total calories for each recipe, per cup, were calculated by using tables of food composition (Gopalan *et al*, 1985). For a few other recipes, the nutritive value was obtained from two publications from Central Food Technology and Research Institute (1976) and National Institute of Nutrition (N.I.N. 1984). The cholesterol content of foods consumed was calculated by using the data given by Swaminathan (1974) and Robinson (1967).

The amount of oil/fat used to prepare the day's menu and ghee used at the table as recorded by each subject was estimated by the investigator. The amount of oil mentioned by each subject (in terms of the steel cup or teaspoons per day) was tallied with the laboratory estimate to check the accuracy of the amount mentioned by the subject. The tally was good. The amount of ghee used was mentioned in terms of number of teaspoons per day. For the amounts of oils, ghee, dalda, milk as well as other flesh foods like mutton, liver, etc., the saturated fatty acid and polyunsaturated fatty acid content was calculated by using the data given by Swaminathan, (1974); NIN (1985) and Gotto and Scott (1973).

With regard to the fat/oil consumed by the students, the information about the quality and total quantity of oil used per recipe was obtained from the hostel mess. The per capita oil in a standard cup serving of the recipe was calculated. Only fixed quantity of flesh foods (usually chicken) are served to each student in a standard container (or cup). This was converted into the standard steel cup measure by the investigator. Sometimes, when the student had consumed extra servings, it was mentioned in the diet sheet. From this, the amount of flesh foods consumed was calculated.

The sugar consumption was calculated from the number of cups of coffee, tea, milk or other beverages used multiplied by the number of spoons of sugar used per cup. Each cup of coffee or tea had around 10 grams of sugar.

The total nutrients provided by the diets for three days were calculated separately for each subject and the average nutrient intake per day was then computed. Like this, the nutrient composition of the diet for 300 subjects was assessed. The data was tabulated before it was subjected to statistical analysis.

2.7 Obesity Index

The standing height and weight of the subjects were recorded using standard equipment, when they reported at the Health Centre to give the blood samples. The obesity index was calculated using the formula weight/height^2 ((Kg/mt^2) given by Norgan and Feerolizzi (1982). On the basis of the index, the subjects could be graded as follows:

Grade		*Index (Wt/ht^2)*
Grade O	...	20.0 – 24.9
Grade I	...	25.0 – 29.9
Grade II	...	30.0 – 40.0
Grade III	...	>40.0

2.8 Blood Pressure Values

The systolic and diastolic blood pressure of each subject was recorded using a "Sphygmo manometer". The measurements were monitored by the medical officer at the University Health Centre. The blood pressure was recorded in the upper arm of the right hand as the subject sat in a chair in a relaxed state for some time.

2.9 Cigarette Smoking

To get information on this, the smoking questionnaire given in the Monograph Series No. 56 on Cardio-Vascular Survey methods (Rose and Blackburn, 1968) was used (vide appendix). Using this questionnaire, the smoking history, the number of cigarettes smoked per day and the extent of inhalation of smoke were recorded. But only the number of cigarettes smoked per day was used in the analysis.

2.10 Family History

To obtain information about the family history, the questionnaires given in the monograph No. 56 (Rose and Blackburn, 1968) were used. The questionnaire is presented in appendix. The information with regard to the incidence of and death due to heart disease in the family (mother, father, brothers, sisters and relatives) was recorded. If any of the parents or brothers or sisters of the individual subject has had a heart attack (Myocardial infarction), the subject was deemed to have a positive family history of heart disease.

2.11 Medical History

To obtain clinical data of subjects, the medical history forms given in Annexure vi and vii of the London School of Hygiene's Cardio-Vascular Questionnaire (Rose and Blackburn, 1968) were used. Annexure vi deals with chest pain on effort, possible infarction and intermittent claudication. Annexure vii seeks information on Dyspnoea. These two questionnaire forms are given in the appendix.

2.12 Bio-Data

The subject's bio-data namely, his name, address, date of birth (age) and other personal particulars were recorded using a schedule for the purpose.

2.13 Coronary Prone Behaviour

Coronary prone behaviour was measured by the Jenkins Activity Survey (JAS) Form C (Jenkins, 1979). This is a self-report, multiple choice questionnaire of 52 items designed to measure the behaviour pattern found to be associated with the risk of coronary heart disease. Form C is the fifth edition of the JAS and the first to be published for the scientific community. It was developed to duplicate the clinical assessment of type A behaviour by the use of standard Psychometric procedures and to make type A assessment accessible to both individual practitioners and to researchers conducting epidemiological studies. The JAS is scored on four scales; the Type–A Coronary Prone behaviour pattern and three fractionally independent components viz., Speed and Impatience (Factor S), Job involvement (Factor J) and Hard-driving and competitive (Factor H).

For each item that contributes to a scale score, each response alternative is assigned numerical points. The scoring was done by hand as per the scoring instructions. The instrument was revalidated on a sample of 30 myocardial infarction patients and 30 normals at Tirupati. All the four scales differentiated the clinical and normal groups at the 0.001 level. A retest reliability co-efficient was calculated which was found to be 0.94 for the composite scale (Jamuna and Sujatha Ramamurti, 1984).

The data recorded with regard to all the risk factors mentioned above was tabulated. It was subjected to a multiple regression analysis (stepwise) in a computer to assess the contribution of several risk factors (viz., Family history. Blood pressure, Height/weight* index, Cigarettes smoked, and dietary components) to variations in serum cholesterol and serum triglyceride levels among the subjects.

RESULTS AND DICUSSION

The findings of the study are presented in three sections, viz. 3.1 Means of Serum Cholesterol (SC), Serum Triglyceridt (STG) and other independent variables for the various age groups; 3.2 Individual contributions of the independene variables viz., Family History, Smoking, Coronary Prone Behaviour, Hypertension, Obesity and Dietary Constituents to the variance in the dependent variables SC and STG; 3.3 An assessment of the step-wise contribution (MRA) of the independent variables to the variance in the dependent variables (SC and STG) in each of the three age groups viz., 20 - 29, 30 - 39 and 40 - 55,

Group differences were analysed using means and *t* tests. For assessing the contribution of independent variables, singly and in combination, to the dependent variables, the Multiple Regression Analysis (MRA) technique was applied. Strict assumption of normality of distribution of dependent variable scores and equal variances in dependent variable at each independent variable point are not needed to calculate correlation and regression measures (Mc Nemar, 1960). However, one has to pause and think of assumptions only when we make inferences from a sample to population. In general it is safe to say that we can ordinarily go ahead with analysis of variance and MRA without worrying too much about assumptions (Kerlinger and Pedhazur, 1973). In the present instance the distribution of the dependent variables viz., SC and STG were more or less normal. Thus, it was felt that there was no need to apply further tests of normality and homogeneity of variance.

As a first step in the analysis, the distribution of the dependent variables and the means of the independent variables are presented.

3.1 The Means for SC, STG and Other Independent Variables for the Various Age Groups

Firstly, the distribution of serum cholesterol values in the 3 age groups was examined. This data is given in Table 14.

Table : 14 Frequency Distribution of Serum Cholesterol Levels and Means for the Various Age Groups

SC mg/dl	Age Groups		
	20 – 29 *f*	30 – 39 *f*	40 – 55 *f*
100 – 125	1	0	0
126 – 150	3	0	0
151 – 175	8	0	0
176 – 200	22	7	3
201 – 225	19	16	8
226 – 250	19	17	17
251 – 275	12	15	17
276 – 300	13	15	18
301 – 325	1	17	22
326 – 350	2	8	10
351 – 375	0	4	4
376 – 400	0	1	1
Total N	100	100	100
Mean	221.47	274.87	280.02
SEm	4.62	5.10	4.43
t value	7.04** (1–2)	0.69** (2–3)	9.13** (1–3)

*Not significant
**Significant at 0.01 level.

The mean difference between the 20 – 29 and the 30 – 39 age groups was statistically significant at 0.01 level, while the difference between the 30 – 39 and the 40 – 55 age groups was not significant. Thus, there was a significant increase in SC values from the 20 – 29 to the 30 – 39 age groups, while the increase from the 30 – 39 to the 40 – 55 age group was insignificant. A further examination of the data in Table 14 revealed that there was a shift in the frequency distribution towards higher values from the lower to the higher age groups.

The frequency distribution and the mean Serum Triglyceride (STG) values in the three age groups are given in Table 15. The mean STG values increased from the lower to the higher age groups and the differences were statistically significant at the 0.01 level. Here too, the frequency distribution was such that there was a shift towards higher values from younger to the older age groups. For eg., 70 per eent of the subjects in the 20 – 29 age group had STG values below 160 mg/dl: only 37 per cent in the 30 – 39 age group and 25 per cent in the 40 – 55 age group had STG values below 160 mg/dl.

The mean SC values and significance of difference among means of normals, smokers and those with positive family history are given in Table 16. In each age group thirty subjects were smokers and thirty had positive family history. The SC values in normals and in those with positive family history increased from the 20 - 29 to the 30 – 39 and the 40 – 55 age groups. Among smokers, SC values increased from the 20 – 29 to the 30 - 39 age group, but not from the 30 – 39 to the 40 - 55 age group. Thus, by and large there were higher values for the older age groups irrespective of whether they were normals, smokers or had positive family history. A similar trend was observed for STG values (Table 17).

Comparing the SC values of normals with those of smokers and with those having positive family history. the values of smokers were higher than that of normals. The values of those with positive family history were also higher than those of normals. These differences were statistically significant at the

Table : 15 Frequency Distribution of Serum Triglyceride Levels and Means for the Various Age Groups

STG mg/dl	Age Groups 20 – 29 *f*	30 – 39 *f*	40 – 55 *f*
40 – 70	7	3	4
71 – 100	20	5	2
101 – 130	23	13	6
131 – 160	20	16	13
161 – 190	11	14	10
191 – 220	12	18	17
221 – 250	3	4	10
251 – 280	0	12	12
281 – 310	2	10	7
311 – 340	1	2	6
341 – 370	1	2	8
371 – 400	0	1	5
Total	100	100	100
Mean	140.59	193.95	226.49
SE$_m$	5.8	7.4	8.4
t value	5.68** (1–2)	2.91** (2–3)	8.43** (1–3)

**Significant at 0.01 level.

0.01 level. However, there were no large differences in mean SC values between smokers and those with positive family history. A similar trend was observed for STG values (Table 17).

Table : 16 Mean Serum Cholesterol Values and Significance of Difference Among Means of Normals, Smokers and those with Positive Family History

S. No.	Sample	20–29 Mean SC Value	20–29 S.D,	20–29 *t* value	30–39 Mean SC Value	30–39 S.D.	30–39 *t* value	40–55 Mean SC Value	40–55 S.D,	40–55 *t* value
1.	Normals	188. (N=53)	24	15** (1–2)	237 (N=44)	36	10** (1–2)	251 (N=45)	35	13** (1–2)
2.	Smokers	263.35 (N=30)	42	1.0@ (2–3)	304 (N=30)	54	1.23@ (2–3)	303 (N=30)	24	1.41 (2–3)
3.	Positive Family History	270 (N=30)	39	18** (1–3)	324 (N=30)	57	13** (1–3)	334 (N=30)	23	20* (1–3)

**Significant at 0.01 level.
@Not Significant.

positive family history were significantly higher than those of normals. These results indicate that smoking and positive family history increase SC values significantly.

The SC values reported by Pinto *et al* (1970) amongst Indians, irrespective of age, sex, income, region, etc., were between 150 and 175 mg/dl. But this study by Pinto *et al* was reported two and a half decades ago (1970). Over these years, the life styles of people have undergone remarkable changes. The stresses of modern living have taken their toll. At the same time, the habits of eating, smoking and drinking have also changed, with some going in the wrong direction. Consumption of refined foods, sweets, variety of baked products (some with plenty of icing), aerated drinks, excess coffee, non-vegetarian food, foods rich with hydrogenated fat like vanaspati (dalda) and butter have increased substantially. Bread, butter, jam and omelettes are frequently substituted for other traditional time-consuming breakfast preparations. This is especially true of those in an urban setting and those in white-collared jobs. Today, whether it is a business transaction or an academic discussion, doing it over a cup of coffee is a common feature. So it is with cigarette smoking. As such, the use of both these have increased.

One consequence of all these significant changes in life styles is bound to be on the blood lipids, pushing their values upwards even among the apparently normal, healthy populations. In the result, these values are not as low as have been reported in some earlier Indian studies.

According to a recent ICMR bulletin (1986), the SC values of apparently healthy adults of the affluent sections in India, were quite high Singh *et al* (1980), reported a high percentage (52.5) of their subjects, who were students between 20 and 22 years, having cholesterol values of 110 – 240 mg/dl. If these men had values ranging from 210 – 240 mg/dl even in their early twenties, what would their values be, when the men reach their thirties and fifties? With the operation of coronary risk factors like hypertension, obesity, stress, positive family history and smoking, in addition to diet, each exerting its own share to the

future risk by elevatting the lipid level in the blood, the cumulative effect of all these risk factors would naturally push up the lipid values gradually over the years. This sounds as a reasonable explanation for the slightly higher mean SC values of 231 7 mg/dl observed in the 30 – 39 and 40 – 55 age groups in the present study. The large number of subjects in these two age groups with SC values 300 mg/dl (Table 14) may also be due to the total effect of various risk factors over a period of time (10 years or 15 years).

The higher the SC levels, the higher is the incidence of IHD (Kannel, 1977[b]), a fact demonstrated too often. Therefore, there is a good possibility that the raised SC levels obtained in this investigation for the apparently normal individuals may, in due course, contribute to the clinical manifesations. The Framingham study had conveyed this point very convincingly in the follow-up reports. Unfortunately, atherosclerosis is a silent and asymptomatic process. One does not know that he is a coronary victim until the clinical manifestations surface, sometimes with telling effect.

Women are supposed to be protected from IHD until they reach menopause and therefore they are shown to have lower SC levels, i e, < 180 mg per cent. But in a study (Devadas *et al*, 1980) conducted on Tamilian and Gujarati women of 20 – 45 years, the mean cholesterol levels reported were 225 and 226 mg/dl. Now, with all the hormonal protection, if women show mean SC levels of 225 mg/dl, one can imagine what it would be in men, when maleness is considered a risk factor.

The levels of STG in the present study for the normal subjects of 20–29 and 30–39 age groups i e, 126.8 and 149.1 mg per cent respectively were within the normal ranges reported by Indian and Western authors (Barrington *et al*, 1980; Gandhi, 1982; Martin *et al*, 1981; Rifkind and Segal, 1983). However, for the 40 – 55 age group, the mean value of 24) mg per cent was some what high compared to Western values. The STG values of Indians are likely to be higher, as the Indian diets are

predominantly cereal based (Gandhi, 1982). A higher percentage i.e. 60–70 per cent of total calories are obtained from carbohydrates. A high carbohydrate diet has been shown to enhance substantially the STG level (Albrink, 1973; Liu *et al*, 1983. This effect might have been prominent in the 40–55 age group, probably being favoured by the other risk factors.

In view of the paucity of Indian data on STG levels, both among normals as well as in other conditions, more studies are needed for establishment of norms. This becomes all the more important in the light of the observation of Barrington *et al* (1980) that STG values and pre-B lipoprotein levels are far higher in the Ischemic Heart Disease patients compared to those of the controls. It is also noteworthy that in the present study, smokers and subjects with positive family history had significantly higher STG levels.

3.1.2 Means for Obesity Index, Blood Pressure and Coronary Prone Behaviour

The obesity indices ranged from 14.5 to 31.7. The mean values were 19.7, 22.5 and 22.8 for the 20–29, 30–39 and the 40–55 age groups respectively. The mean obesity indices for the three age groups indicated that none of them fell into the obese categories. The small SEm values (0.26, 0.27 and 0.26 respectively) suggest that there was not much variation in the obesity indices. Further, the differences among the age groups in the obesity indices were not significant, though there was an increasing trend from the lower to the higher age groups.

The mean blood pressure values for the three age groups showed slight increases with age in both systolic and diastolic. The mean systolic blood pressure values for the 20 29, 30–39 and 40–55 age groups were 122.31 (110–128), 126.03 (120–140) and 128.52 (114–140) mm Hg respectively and are within the normal range. The mean diastolic blood pressure values viz., 81.06 (78–90), 84.24 (70–94) and 87.70 (70–98) mm Hg for the 20–29, 30–39 and 40–55 age groups respectively were also within the permissible range for normals. The small differences among the age groups, however, were statistically significant.

Table : 18 Mean and SE_m of Coronary Prone Behaviour Sub-Scores for the Various Age Groups

S. No.	Age Group	Type A $Mean_m$	Type A SE_m	Type A *t* value	Speed & Impatience Mean	Speed & Impatience SE_m	Speed & Impatience *t* value	Job Involvement Mean	Job Involvement SE_m	Job Involvement *t* value	Hard Driving Mean	Hard Driving SE_m	Hard Driving *t* value
1.	20–29	154	4.4	4.96** (1–2)	98	3.8	4.25** (1–2)	85	2.4	8.60** (1–2)	86	3.2	6 93** (1–2)
2.	30–39	185	4.5	1.23 (2–3)	121	3.7	3.15** (2–3)	127	4.3	3.00** (2–3)	114	2.8	1.65* (2–3)
3.	40–55	176	4.8	3.55** (1–3)	105	3.5	1.32 (1–3)	111	3.3	6.49** (1–3)	108	2.7	5.42** (1–3)

*Significant at 0.05 level

**Significant at 0.01 level.

The mean scores for coronary prone behaviour (JAS) for the three age groups (Table 18) showed an interesting trend. They increased from 20–29 to the 30–39 age group and then showed a decrease in the 40–55 age group. This was true of all the sub-scores as well. Most of the differences were statistically significant. Obviously, the 30–39 age groups showed the highest scores. This indicates that job involvement, hard driving and speed and impatience were high in the 30–39 age group and 40–55 age group compared to that of the 20–29 age groups, where these scores were comparatively low. To what extent, these behaviours are environmentally induced or whether the heightened manifestation of these behaviours in the 30–39 age period reflects developmental (stage) life styles are questions that cannot be easily answered. They need to be investigated.

3.1.3 Means of Dietary Variables

The mean values for total calories, carbohydrates and protein for the three age groups are given in Table 19. The mean values of total calorie content compare well with Recommended Dietary Allowances (RDA) of ICMR (1984) 2400 and 2800 for men of sedantary and of moderate activity respectively. The RDA given by ICMR included added provisions over and above the actually determined values to ensure good health. As such, the calorie intake above the actual requirement in sedantary people would contribute to positive energy balance. When the ranges are examined, it is evident that there is considerable variation from individual to individual in the sample. If the total calorie content is more than optimal, it contributes to positive energy balance and thus to over-weight. Over-weight and obesity enhance the lipid synthesis and affect the heart of an individual in many other ways. Therefore, as per the dietary recommendations the very first recommendation is consideration of a calorie allowance that would maintain the ideal body weight of an individual.

Nearly 59 per cent of the total calories were obtained from carbohydrates by the subjects in the sample. The percentage of calories from carbohydrates in the Western diets is around 40 per

Table 19 Mean and SE_m for Total Calories, Carbohydrates and Protein for the Various Age Groups

Sl. No.	Age Group	Calories			Carbohydrates (gms)			Protein (gms)		
		Mean	SE_m	*t* value	Mean	SE_m	*t* value	Mean	SE_m	*t* value
1.	20–29	2436 (1813–3634)	32.09	0.32 (1–2)	330 (190–448)	5.2	0.68 (1–2)	75.4	1.31	1.09 (1–2)
2.	30–39	2426 (1543–3442)	38.10	2.50** (2–3)	336 (196–526)	7.2	0.59 (2–3)	74.2	1.50	4.93** (2–3)
3.	40–55	2289 (1728–3042)	35.80	3.06** (1–3)	330 (188–525)	7.3	0 (1–3)	64.0	1.43	5.88** (1–3)

**Significant at 0.01 level.

cent. When compared against this figure, the contribution of carbohydrate as a source of energy is rather high in the present investigation. Though carbohydrate generally has not been shown to affect the SC values much, some studies have shown that the STG values go up significantly when carbohydrate supplies 60 per cent or more of calories (Liu *et al*, 1983). Albrink (1973) pointed out that with 75 per cent calories from carbohydrates, there could be universal increase in TG, perhaps permanent in susceptible individuals. The higher amount of carbohydrate, esspecially if it includes high amount of sugar, can contribute to hyper triglyceredimia (Hayford *et al*, 1979; Yudkin, 1972). But, if the fibre is enhanced (i.e. tripled) the high carbohydrate diet does not elevate TG levels (Lewis *et al*, 1981).

The higher amounts of protein intake were, in many cases, due to the consumption of non-vegetarian items like chicken and mutton. Among vegetarians, the major sources for protein were pulses and milk. The mean values of protein intake were higher than the RDA of 55 g protein per day.

The values for fat, P/S ratio and cholesterol are given in Table 20. The range of values for fat were from 43.1 to 99.9 g per day. For the three age groups, the mean values were 74.60, 74.64 and 69.86 g. In the 20–29 and 30–39 age groups, 29.3 per cent of the total calories and in 40–55 age group, 27.4 per cent of the total calories were obtained through fat. The American Heart Association (A.H.A.) advocated that the fat calories in the preventive diet are to be restricted to about 30 per cent (Muller, 1973) As compared to this, the total calories from fat were almost optimal in the sample of the present study.

The SFA ranged from 2.8 to 32.4 and PUSFA ranged from 3.8 to 33.0 g in the sample. With regard to the P/S ratio, the mean values were 1.15 (0.3–2.0), 1.13 (0.4–1.7) and 1.33 (0.6–6.5) for the three age groups respectively. The mean values of saturated fatty acid and polyunsaturated fatty acid content for the three age groups were 12.13 and 12.46; 12.72 and 12.98; and 14.85 and 18.17 g respectively. The grand mean P/S ratio for the entire sample was 1.206. In a preventive diet of A.H.A., the P/S ratio was 1.4:1 or 1.4. As compared to this, P/S ratio

Table 20: Mean and SE_m for Fat, P/S Ratio, and Cholesterol for the Various Age Groups

S. No.	Age Group	Fat Mean	Fat SE_m	Fat *t* value	P/S Mean	P/S SE_m	P/S *t* value	Cholesterol Mean	Cholesterol SE_m	Cholesterol *t* value
1.	20–29	74.60	1.24	0.02@ (1–2)	1.15	.039	.02@ (1–2)	83	1.06	29.33** (1–2)
2.	30–39	74.64	1.45	2.29* (2–3)	1.13	.038	.41@ (2–3)	127	1.05	3.12** (2–3)
3.	40–55	69.16	1.50	2.49** (1–3)	1.33	.074	.48@ (1–3)	132	1.20	3.62** (1–3)

@Not Significant
*Significant at 0.05 level
**Significant at 0.01 level.

obtained for the sample in the present study was optimal. The polyunsaturated fatty acid was mostly obtained from the vegetable oils, groundnut oil being the most commonly used cooking oil in this region. In the 40–45 age group two subjects recorded in the diet questionnaire, that they were using gingelly oil and two subjects sunola and saffola oils. Their P/S ratios were quite high, around 5 and 6. The P/S ratio of the third age group subjects was 1.33, slightly higher than the values of 1.15 and 1.13 observed for the 20–29 and 30–39 age groups respectively. The saturated fatty acid content was mostly from ghee, dalda and milk fat and for the non-vegetarians it came from mutton in addition. About 5 per cent of the total calories were obtained from SFA and about 5 per cent in the 20–29 and the 30–39 age groups and 7 per cent in the 40–55 age group came from PUSFA. Therefore, it could be said that the quantity and quality of fat consumed by the subjects in the sample was more or less optimal.

The mean dietary cholesterol values were 83 (4–411), 127 (4–370) and 132 (4–417) mg in the age groups 20–29, 30–39 and 40–55 respectively. There was an increase in the values from younger to older age groups. This increase was statistically significant. The dietary cholesterol values in this study were much lower when compared to the mean dietary cholesterol content of a Western diet which is 500–600 mg/day. The A.H.A., recommended that cholesterol be restricted to <300 mg/day in a preventive diet. As compared with these values, the mean dietary cholesterol content was much lower in the present study.

There is quite a controversy regarding serum cholesterol response to dietary cholesterol. A linear and positive relationship was shown in some studies, but addition of 3–6 eggs in a day did not elevate SC in some other studies. It is postulated that there is a threshould level upto which, the dietary cholesterol affects the SC value but above this, there is no change. This upper-limit could be 250 or 400 mg (Dawber *et al*, 1982). Like-wise there is a wide variability in the individual responses to dietary cholesterol. The individuals are termed as hyporesponders and hyperresponders depending on their sensitivity in responding to dietary cholesterol values. Some investigators had stated that it

is the interdependability of dietary fat and cholesterol which exerts maximum effect on SC values (eg. Muller, 1973; Williams *et al*, 1985; Angelico, 1985). A high P/S ratio with high PUSFA, did not elevate SC values inspite of a high dietary cholesterol content (Nut. Rev., 1985).

Thus, it is likely that in the present study the variations in the dietary fat and dietary cholesterol content among individuals might have affected the SC values. For example, a mutton preparation or an egg preparation with dalda (a hydrogenated fat) or ghee can increase the SC values with its high cholesterol and high SFA content. Of course, the genetic susceptibility of an individual may determine the quantum of response. Thus it is the interplay of a combination of factors which ultimately affects the role of a particular variable.

The mean dietary values of Vit. C, fiber and sugar for the three age groups are presented in Table 21. The mean Vitamin C values of 41.3, 45.2 and 44.5 mg for the three age groups compare favourably with 40 mg, which is RDA. The range of values was 8 to 138; 17 to 261 and 17 to 120 mg per day in the three age groups respectively.

The dietary fiber, which is three times the crude fiber content (approximately) is difficult to analyse accurately. Recently the dietary fiber content of Western foods was given (Lanze and Butrum, 1986). But for Indian foods, only crude fiber data is available. The mean crude fiber content of diet was 4.8, 5.2 and 5.1 g for the 3 age groups respectively. The values ranged from 1 g to 9.9 g per day for the entire sample. Though various components of fibre like pectin, guar gums etc. have been shown to exert effects differently on lipids, yet only total crude fiber content was calculated as data is available only on this. A high fiber content decreases the serum lipid levels by enhancing the excretion of neutral steroids and bile acid synthesis. Vitamin C and fiber affect the metabolism of cholesterol, by increasing the catabolism of cholesterol. They decrease the blood lipid levels and thus are inversely related.

Table: 21 Mean and SE_m for Vitamin C, Fiber and Sugar for the Various Age Groups

S. No.	Age Group	Vitamin C Mean	Vitamin C SE_m	Vitamin C t value	Fiber Mean	Fiber SE_m	Fiber t value	Sugar Mean	Sugar SE_m	Sugar t value
1.	20–29	41.3	0.11	12.6** (1–2)	4.8	1.44	0.19@ (1–2)	14.4	.075	13.5** (1–2)
2.	30–39	45.2	0.29	8.94** (2–3)	5.2	1.80	0.03@ (2–3)	25.8	.144	2.01* (2–3)
3.	40–55	44.5	0.22	13.3** (1–3)	5.1	1.64	0.11@ (1–3)	27.1	.154	14.5** (1–3)

**Significant at 0.01 level
*Significant at 0.05 level
@Not Significant.

The average sugar consumption ranged from 6 to 55 g per day for the sample. The mean values were 14.4, 25.8 and 27.1 g/day in the 20-29, 30-39 and 40–55 age groups respectively. The mean sugar consumption increased from the 20–29 to the 40–55 age group. The higher sucrose content of the diet has been shown to elevate the triglyceride levels in the blood. In a recent study (Nut. Rev., 1985), it was stated that catecholamine release is also enhanced by dietary carbohydrate, especially the sucrose component of it. The catecholamines mobilise the free fatty acids from the adipose tissue. The triglyceride synthesis (endogenous) from the free fatty acids goes up as a consequence.

The Obesity Indices and the mean systolic and Diastolic blood pressure readings obtained in this study were by and large within normal limits in all the three age groups. Indian standards to compare values for type A Behaviour Pattern were not available.

The mean nutrient composition of the diets consumed by the subjects of the present sample was within normal limits of the Recommended Dietary Allowances (RDA). In brief, the facts that have become salient in the foregoing were that the SC and STG levels of the age groups 30–39 and 40–55 were moderately high. This needs to be handled since high blood lipid values have been consistently reported to be strongly associated with IHD.

By far the most comprehensive and significant consensus on the relationship of blood cholesterol to IHD was reported in the National Institute of Health Consensus Development Conference (NIHCDC) statement. It stated in unmistakable terms that a large body of evidence of many kinds links elevated blood cholesterol levels to Coronary Heart Disease (CHD). Regarding the role of other factors, it adds that cigarette and high blood pressure have been identified as strongly associated with CHD, in addition to high blood cholesterol levels. Risk is greater in men, increases with age, and has a strong genetic component. Obesity, physical inactivity and behaviour pattern are also risk factors.

The panel of the NIHCDS concluded that it has been established beyond reasonable doubt that lowering definitely elevated blood cholesterol levels will reduce the risk of heart attacks caused by CHD. For this, it recommended that subjects with high blood cholesterol levels (values above the 75th percentile) should be treated intensively by dietary means under the guidance of a physician or a dietitian. In addition, the recommendation states that people should be advised to adopt a diet that reduces total dietary fat intake to 30 per cent of total calories, reduces saturated fat intake to less than ten per cent of total calories, increases polyunsaturated fatty acid intake to no more than ten per cent of total calories and reduces daily cholesterol intake to 300 mg or less (NIH Consensus Development Conference Statement, 1985).

Gotto (1985) in his individual report stated that persons with plasma cholesterol levels above 240 mg/dl should be carefully evaluated and should change their diet to reduce their plasma cholesterol levels. He added that a low fat diet designed to lower plasma cholesterol levels slowed or stopped the growth of plaques in many patients. In the context of the foregoing, the role of diet in influencing the blood levels of cholesterol, becomes important. These observations considered in the light of the findings of the present study, viz., the mean cholesterol levels of the 30–39 and 40–55 age groups were 274.87 and 280.2 mg/dl, take on added significance.

Serum triglycerides have also been reported to be an independent additional risk for the development of coronary disease. The rate of coronary disease increased linearly with increasing triglycerides or cholesterol levels. The combined elevation of both cholesterol and triglyceride carry the highest risk (Tzagournis, 1978).

Albrink (1973) stated that hypertriglyceredemia may contribute to atherosclerotic cardio-vascular disease by mechanisms independent of atheroma formation by causing symptomatic expression as angina pectoris or as sudden death, through

the mechanism of action is still not very clear. The association between elevated triglyceride levels and Ischemic Heart Disease are also not established with the same certainty as with cholesterol levels. The focus on triglyceride as a risk factor in IHD is only in the recent past. Weight reduction, abstention from sucrose and alcohol and a restricted carbohydrate diet or a moderate carbohydrate diet with more fiber in it, can bring down the elevated triglyceride levels effectively. Controlling hyperlipidemia, elevated cholesterol and elevated triglyceride levels is the most important goal in treatment and in preventive trials and has gained importance as a strategy in the public health programmes of the West.

5.2 Individual Contributions of the Independent Variables to the Variance in the Dependent Variables

The focus of the present study was to assess the contribution of the independent variables singly and in combination, to the variance in the dependent variables viz., serum cholesterol (SC) and serum triglycerides (STG). The multiple regression analysis (MRA) as a technique seeks to identify and estimate the magnitude of the variance in the dependent variable (in this case the SC and STG) that is shared by several independent variables. In other words, the effort here is directed towards explaining a single phenomenon which is complex and is of multiple etiology (Cohen, 1968).

Generally speaking, MRA is an appropriate method of analysis when (a) the dependent variable is continuous; (b) when the independent variables are both continuous and categorical; (c) when cel frequencies in a factorial design are unequal and disproportionate; and (d) when studying trends in data. Since the variables employed in the present investigation fit into conditions described above, MRA appeared to be a suitable method of analysis. Moreover, MRA is often the best method of analysis on non experimental data in which there are several independent variables. No matter what the scales of measurement are, or what the kind of variable is, useful analysis

can be done and interpretations made by using MRA. A final strength of MRA is its rich yield of various statistics to be used in the interpretation of data (Kerlinger and Pedhazur, 1973).

3.2.1 Individual Contributions of Independent Variables

MRA was carried out independently for SC and STG to see how the independent variables vary in their contribution to the variance in each of the two dependent variables viz., SC and STG. For this purpose, MRA was carried out with the help of an available computer programm (Kerlinger and Pedhazur, 1973). The first output of the MRA that was used was the R^2 values of each of the independent variables (treated separately) pertaining to the first age group (20–29). The R^2 values show the variance shared in common by each one of the independent variables and the dependent variable (SC and STG) separately. The R can be interpreted much like an ordinary co-efficient of correlation. R^2 is called the co efficient of determination.

The per cent variance contributed by each one of the independent variables to variance in SC and STG in the 20–29 age group was assessed. It is seen that smoking contributed the maximum to the variance in SC values (52.4 per cent). This was followed by family history (39.3 per cent), Type A Behaviour (29.16 per cent), Job Involvement (12.6 per cent), Speed and Impatience (11.6 per cent), Hard Driving (9.8 per cent), Age (8.6 per cent), P/S (4.16 per cent), Obesity Index (1.74 per cent), Protein (1.25 per cent) and Fat (1.8 per cent) in that order. The contributions of the other independent variables were very low, perhaps insignificant. In other words, the major contributants to the variation in SC were smoking, family history, Type A Behaviour, job involvement, speed and impatience, hard driving and age.

In so far as STG values were concerned, the major contributions to its variation in the 20–29 age group were, Family history (16.72 per cent), Smoking (7.34 per cent), Type A Behaviour (7.08 per cent), Speed and Impatience (6.66 per cent), Obesity Index (6.15 per cent) and Hard Driving (5.06) in that order. The contribution of other variables were of minor order.

The contribution of the Independent Variables to the variance in the SC and STG values in the 30–39 age group indicates that Family History accounted for the maximum of the variance in SC values (35.64 per cent). This was followed by Type A behaviour, (22.08 per cent), Smoking (13.76 per cent), Hard Driving (8.82 per cent), Job Involvement (4.93 per cent), Speed and Impatience (4.84 per cent), P/S ratio (4.47 per cent) PUSFA (4.24 per cent) and Cholesterol (2.53 per cent) in that order. The contribution of other variables was not significant. Thus, the major portion of the variance was accounted by Family History, Type A Behaviour and Smoking.

The important determiners of the variation in the STG in the 30–39 age gronp were Family History (17.22 per cent), Smoking (14.36 per cent), Type A Behaviour (12.25 per cent), Age (5.85 per cent), Hard Driving (5.2 per cent), Saturated Fatty acid (2.96 per cent), Polyunsaturated Fatty acid (2.72 per cent). The contributions of the remaining variables were insignificant.

In the 40–55 age group, Family History again contributed the maximum (45.02 per cent) to the variance in SC values, followed by Smoking (19.9 per cent), Type A Behaviour (19.36 per cent), Job Involvement (10.11 per cent), Speed and Impatience (9.67 per cent), Diastolic B.P. (7.9 per cent), Hard Driving (7.72 per cent), Vitamin C (5.06 per cent), Systolic B.P. (4.9 per cent) and Fiber (3.96 per cent) leaving the other variables insignificant.

Regarding the contributions of independent variables to the variance in the STG values in the 40–55 age group, Obesity Index (15.21) contributed the maximum followed by Hard Driving Behaviour (9.81), Job Involvent (7.67 per cent), Type A Behaviour (5.96 per cent), Family History (5.8 per cent), Sugar (3.6 per cent), P/S Ratio (3.57 per cent), Speed and Impatience (3.31 per cent) Smoking (3.03 per cent) in that order.

Subsequently, the contribution of independent variables to the dependent variables SC and STG in the whole group of 300 subjects (i.e. the group considered as one unit) was assessed. The percent variance accounted by each factor is given in table 22.

Table: 22 Individual Contribution of the Independent Variables to the Variance in the Serum Cholesterol and Serum Triglyceride Values in the 20–55 Age Group

S. No.	Variable	Serum Cholesterol Percent of Variance Accounted	Serum Triglycerides Percent of Variance Accounted
1.	Age	16.73**	18.92**
2.	Obesity Index	9.18**	8.82**
3.	Systolic B.P.	2.59*	2.75*
4.	Diastolic B.P.	7.56**	5.11*
5.	Smoking	22.27**	7.56**
6.	Family History	30.14**	8.94**
7.	Type A	27.71**	9.49**
8.	Speed & Impatience	10.24**	3.38*
9.	Job Involvement	16.16**	11.15**
10.	Hard Driving	17.14**	12.53**
11.	Calories	0.10	0.61
12.	CHO	0.07	0.42
13.	Protein	0.12	1.88
14.	Fat	0.18	0.32
15.	SFA	1.42	1.46
16.	PUSFA	2.89	4.20*
17.	P/SF	0.004	1.64
18.	Cholesterol	2.54*	0.79
19.	Vitamin C	0.07	0.004
20.	Fiber	0.014	0.006
21.	Sugar	2.92	5.79*

*Significant at 0.05 level

**Significant at 0.01 level

In as much as the three age groups were combined into one unit, the order of importance of variables is likely to change. However Family History remained the chief determiner of SC values (30.14 per cent) followed by Type A Behaviour (27.77 per cent), Smoking (22.27 per cent), Hard Driving (17.14 per cent), Age (16.83 per cent), Job Involvement (16.16 per cent), Speed and Impatience (10.24 per cent), Obesity Index (9.18 per cent), Diastolic B P. (7.56 per cent), Sugar (2.92 per cent), PUSFA (2.89 per cent), Systolic B.P. (2.59 per cent) and dietary cholesterol (2.34 per cent). Among these variables, Family History, Type A Behaviour. Smoking, Hard Driving. Age, Job Involvement, Speed and Impatience and Obesity Index can be regarded as the important ones.

With regard to STG, when the group as a whole was considered, Age (18.92 per cent), Hard Driving (12.53 per cent), Job Involvent (11.15 per cent), Type A (9.49 per cent), Family History (8 94 per cent), Obesity Index (8.8 per cent) and Smoking (7.56 per cent) were the important independent variables.

The examination of independent contributions of the different variables to SC levels in this study showed that Family History was an important variable determining cholesterol levels. Of nearly equal significance were Type A Behaviour and Smoking.

Age differences appeared to contribute approximately 17 per cent. Next in order of importance, were the sub-factors of Type A Behaviour namely Hard Driving, Job Involvement and Speed and Impatience. The contributions of other variables (except obesity index and Diastolic B.P. which became significant when the overall group was considered) were not appreciable.

Since family history contributed to the variance in serum lipid levels to a considerable extent, the relationships between the independent and dependent variables in those subjects with positive family history were assessed separately. The data presented in table 23 indicates that the effects of dietary variables become more pronounced and significant in these susceptible subjects. Proteins, fat, especially the quality and dietary cholesterol

influenced the serum cholesterol levels remarkably, whereas calories, carbohydrates, affected the serum triglyceride sugar levels to a greater extent. This fact illustrates the importance of interaction of dietary variables and serum lipid levels in genetically susceptible individuals.

Table: 23 Individual Contribution (co-efficient of Variation) of Independent Variables to Variance in Serum Lipid Levels in Individuals with Positive Family History (N = 60, Age Groups = 20–55 years).

S.No.	Variable	Variance accounted in Serum Cholesterol level	Variance accounted in Serum Triglyceride level
1.	Obesity Index	12.4	11.8
2.	Systolic Blood Pressure	13.1	11.6
3.	Diastolic Blood Pressure	15.2	13.1
4.	Smoking	26.2	13.4
5.	Type A Behaviour	30.4	15.4
6.	Calories	8.8	10.4
7.	Carbohydrate	4.6	12.4
8.	Protein	2.8	7.9
9.	Fat	4.1	3.8
10.	Saturated fatty acid	8.4	6.2
11.	Polyunsaturated fatty acid	9.2	7.1
12.	Cholesterol	8.6	3.5
13.	Vitamin C	3.8	2.6
14.	Fiber	3.2	4.9
15.	Sugar	9.4	11.8

3.3.2.2 **Intrepretation of individual Contributions**

Individual contributions of the independent variables are based on the associations between the dependent variable and each of the independent variables. It does not take into account the other variables. This sort of analysis helps us to assess the relationships of the variables to the dependent variables without taking into account the influence of other variables.

The foregoing statistical analysis of the contribution of the independent variables, taken one at a time- seems to suggest a less important role for the dietary constitutents when compared to Family History, Smoking and Type A Behaviour in determining SC levels. For instance, the analysis of factors in the 20–29 age group showed that no dietary constituent contributed more than two per cent to the variance in SC values. This seems to be true even with regard to the accounting of variation in triglyceride levels of this group. Evidently, dietary constituents did not appear to be playing a major role in determining the SC values, so far as this sample was considered. Some Western studies (eg., The Framingham Study) and some Indian studies (eg., Juneja and Sharma, 1981; Singh *et al*, 1980), have not found any significant relationship between diet and SC levels. Juneja and Sharma (1981) assessed the relation between dietary variables and SC levels among normals and heart patients and stated that diet correlated poorly with SC levels. In a 12 year follow up on women in Sweden Lapidus et al (1986) found no correlation between diet and incidence of CHD. The net effect of dietary variables on reduction in SC levels may be 3–5 per cent (Conference Report, 1982). However, other studies did observe a relationship between dietary constituents and SC levels (vide 1.3).

The subjects in the age group 20–29 years were students messing in hostels which supplied a more or less uniform menu without much variation in the ingredients from meal to meal and person to person. Probably, this did not provide enough scope for the dietary intake to effect an appreciable variation in SC and STG levels among these individuals. It is also seen that the diet

consumed by the hostel residents is not much of an atherogenic diet, as could be surmised from the mean values and ranges for the dietary constituents.

Further, this being an epidemiological study, it was not possible to manipulate the dietary intake and see the effect of such manipulation on SC and STG levels, as was done in some of the experimental studies reporting association between dietary intake and SC and STG levels (Muller, 1973; Berry *et al* 1986; Mattson and Grundy, 1985).

Even in the 30–39 and 40–55 age groups, the individual contributions of dietary constituents to SC levels have not been impressive as compared to the non-dietary risk factors. However, some of the dietary constituents viz., PUSFA, DC, P/S ratio in the 30–39 age group, and protein, Vitamin C, Fiber in the 40–55 age group registered between two and five per cent contribution to the variance in SC levels.

The contributions of dietary constituents to STG levels in these two age groups were also not sizable as compared to the other non-dietary risk factors. However, some contributants viz., SFA, PUSFA, in the 30–39 age group; and Fat, P/S ratio and Sugar in the 40–55 age group determined between two and five per cent of the variance in the STG level. In essence, the examination of individual contributions of the variables under study to SC and STG levels have brought out a significant role for Family History, Smoking and Coronary Prone Behaviour in influencing SC and STG levels. A lesser role, nevertheless appears to exist for some of the dietary constituents in influencing SC and STG levels. Next to Family History and Smoking, Type A Behaviour and its constituents were important contributants to SC and STG levels.

With regard to dietary constituents in the 20–29 age group, the contribution of P/S ratio was significant at the one per cent level. Obesity index and fat content contributions were near the 1 per cent level of significance, though not highly significant. Probably these influences may gain more significance over the years when individuals start putting on weight and the lipid levels go up. Stamler (1983) reported high cholesterol

levels in only those with overweight. Fat, its quality and quantity have been shown in many investigations (vide 1.3.2) to affect Serum Cholesterol levels.

In the 30–39 age group, both P/SF and PUSFA contributed to some extent to variance in SC and SFA in STG levels (at the 0.05 level). This only emphasizes the fact that among the dietary constituents, fat does affect to some extent serum cholesterol and triglyceride values, though Albrink (1973) stated that exogenous fat was never a cause of hypertriglyceridemia.

With regard to the dietary variables in the 40–55 age group, protein had a significant relation (0.06 per cent) to SC levels. Protein in the diet was obtained not only from pulses and milk as in the case of vegetarians, but also from flesh foods. Animal protein, especially mutton was consumed by many subjects who were non-vegetarians in that group. Therefore it is possible that the animal protein, (mutton) might have contributed to this influence on SC levels in this group.

Williams *et al* (1986) reported from their study, that animal protein consumption correlated positively and plant protein consumption correlated negatively with TGs, smaller Low Density Lipoprotein mass and very Low Density Lipoprotein mass. These correlations were significant. They further reported that consumption of plant and animal protein may exert an effect on Lipoprotein concentrations through unmeasured dietary components associated with plant and animal products or through the specific amino acid pattern of various proteins influencing directly serum lipoprotein concentrations. Two amino acids in particular, Arginine (higher in plant protein) and Lysine (higher in animal protein) may influence apolipoprotein E synthesis and this could alter hepatic uptake of remnant lipoproteins. Arginine is a potent stimulator of glucagon synthesis and the glucagon–insulin ratio in plasma has been shown to increase when plant protein is substituted for animal protein in the diet. An increase in this ratio is associated with a decrease in plasma TG and in cholesterol concentration. On the other hand, increase in animal protein contributes to increased cholesterol concentration.

The inverse relationship of Vitamin C and fibre were significant at the 5 per cent level. The hypocholesterolemic property of vitamin C and fiber might have been more pronounced in certain individuals especially consuming higher quantities of vitamin C and those consuming that particular fraction of dietary fiber which can bring down the cholesterol level (i.e. pectin, guar, cellulose etc., which enhance the excretion of neutral steroids). Pectin increases fecal bile acid excretion and fecal steroid excretion, causing increased concentrations of hepatic lipoprotein B/E receptors, so that circulating cholesterol-rich lipoproteins are cleared more rapidly (Williams *et al* 1986). The interaction of various dietary constituents is a complex phenomenon, which cannot be explained easily. As the individual contribution of Vitamin C and fiber are somewhat significant in this analysis, it would be useful that these two dietary constituents i.e., Vitamin C and fiber be consumed satisfactorily either as per RDA or slightly more than that. Fresh vegetables, fruits and green leafy vegetables are also good sources of dietary fiber. To this, if the fiber from whole cereals and legumes is added, it would be beneficial to the individual in maintaining favourable lipid levels.

When the individual contributions of dietary constituents for the whole group (20–55) are examined, sugar consumption had significant ($P<0.05$) relation both with SC and STG level. As already mentioned in the literature (1.3) fructose and sucrose increase lipogenesis. Therefore TG and cholesterol synthesis might have been more and thus might have contributed to the higher levels in some individuals, especially among those in this study with genetic susceptibility.

The next variable significant at the 5 per cent level was PUSFA which affected both SC and STG levels. The beneficial effects of polyunsaturated fatty acid were illustrated in many clinical trials. The retrospective and prospective investigations in which very simple dietary modifications of substituting PUSFA for saturated fatty acids fetched clinically rewarding results, (Ahrens, 1985; Glueck *et al*, 1986; Mattson, 1973). Salonen

and Puska's (1983) findings supported the hypothesis that changes in fat consumption lead to changes not only in SC levels but also in blood pressure.

The role of polyunsaturated fatty acids in lowering cholesterol levels was very well brought out in these studies. Its role in reducing the higher VLDL levels and thus VLDL triglycerides was explained by Beynen and Kalan (1985), as that PUSFA are converted into ketone bodies instead of VLDL triglycerides by liver. Based on this information it is recommended that a higher polyunsaturated fatty acid and a lower saturated fatty acid content in the dietary fat is a desirable goal. Probably, in the long run this may alter the adipose tissue linoleic acid content as was shown in some studies. This would also bring down the higher serum cholesterol and serum triglyceride levels.

In this study the P/S ratio was calculated based on PUSFA and SFA content of the foods, fats and oils. The predominantly used oil by subjects in this study was groundnut oil. The SFA and PUSFA content of groundnut oil was calculated. But, the groundnut oil that was purchased by the subjects in the open market might not have been pure groundnut oil. For the tendency in these parts is to adulterate the material with a cheaper oil. The cheaper oil that is available is palm oil. Palm oil has a very low polyunsaturated fatty acid content and high SFA. When groundnut oil is adulterated with palm oil, naturally the quality and thus the P/S ratio of groundnut oil is also likely to be affected. The effect of PUSFA and thus of P/S ratio would have probably been more marked, if the purity of oils sold in the open market was maintained in a strict manner. Illustration of the effect of quality of fat/oil on blood lipid levels, on a long term basis, needs to be assessed in prospective studies in India. Similarly, the other dietary variables mentioned already should also be investigated for their roles in such experiments, wherein the traditional dietary patterns are simulated but not with extraordinary or unusual combinations such as too high a quantity of fiber or PUSFA or sucrose.

While evaluating the relationship of various dietary constituents to SC and STG levels it is useful to remember that results from epidemiological investigations are only suggestive. Interactions may occur among dietary constituents and relationships may thus become complex and cannot be explained easily unless most of the variables are controlled and metabolic ward type of researches carried out. Nevertheless, the foregoing results and observations do have their own significance.

3.3 Stepwise Contribution of the Independent Variables to the Variance in the Dependent Variables SC and STG

MRA stepwise analysis would indicate the amount of variance additionally accounted for by each of the variables added according to their value of correlation with the dependent variable. The variable to be entered in each step was chosen on the basis of its relative contribution i.e. highest partial correlations. Thus a stepwise solution was pursued to utilise these features to advantage.

3.3.1 Step-wise Contribution of Independent Variables

The step-wise analysis for SC of the 20–29 age group indicated that smoking was entered as the first variable, since it had the highest zero order correlation with SC. It accounted for about 53 per cent of variance in SC. The second variable to be entered was Family History since it had highest partial correlation. With the addition of this variable in the equation, the R^2 value rose to 0.6888, the increase in R^2 was .165 indicating that this variable added 16.5 per cent to variance accounted for, over and above the contribution of Smoking. The third variable entered was Type A Behaviour. It had additionally contributed 4.7 per cent to the variance. Vitamin C was the next variable to be entered and it contributed to 1.1 per cent to the variance additionally. Age, obesity index and PUSFA were the variables to follow, contributing 0.53, 0.56 and 0.58 per cent respectively. The other remaining variables did not make any significant contribution to the SC values additionally. Thus, the data indicated that in the 20–29 age groups, smoking, family

history, Type A Behaviour had together accounted for approximately 74 per cent of the variance suggesting their importance as joint predictors of SC.

Next, a step-wise solution of serum cholesterol values for the 30–39 age group was carried out. In this analysis, family history was the first variable that was entered. It accounted for 35.65 per cent of the variance in SC values. The variables to be entered in succession were Type A Behaviour followed by smoking, vitamin C and carbohydrates in that order. They added 9.11; 3.36; 2.25 and 1.1 per cent respectively. The additional contributions of the remaining variables were less than one per cent each and together they added 3 per cent only (54.41–51.38) to the variance. Therefore in the 30–39 age group the important contributants to SC levels were family history, type A behaviour, smoking and vitamin C jointly accounting 50 per cent of total variance.

In the 40–55 age group, the most important contributor to SC values was Family History which determined 44.99 per cent of variance in SC values. This was followed by Smoking, Type A Behaviour and Diastolic B.P. in that order, contributing 8.38, 3.94 and 2.63 per cent respectively. These variables together determined approximately sixty per cent of variance. The remaining variables together added only 3.48 per cent (63.42–59.94) to the variance in SC values.

Now that the three age groups have been considered separately, the contribution of the different variables to the SC values when the group was treated as a whole (20–55 years) was examined. The data can be seen in Table 24. The MRA – stepwise showed that Family History was the prime variable. It contributed 30.11 per cent to the variance in SC levels. This was followed by the variable age, which added 16.86 per cent. Then came smoking, followed by Type A Behaviour adding 10.04 and 4.92 per cent respectively. Vitamin C and Job Involvement were the next two variables that added .99 and 1.03 per cent respectively. The remaining variables together determined 1.39 per cent of variance (65.35–63.96). The variables Family History, Age, Smoking, and Type A Behaviour together accouted 61.94 per cent of variance.

Table : 24. Summary Table of MRA – Step Wise Solution of Serum Cholesterol Values in the 20–55 Age Group

Step No.	Variable Entered	R^2	Increase in R^2	F Value to Enter or Remove
1.	Family History	.3011	.3011	128.36
2.	Age	.4686	.1686	94.43
3.	Smoking	.5701	.1004	69.16
4.	Type A	.6194	.0492	38.16
5.	Vitamin C	.6292	.0099	7.82
6.	Job Involvement	.6396	.0104	8.41
7.	Obesity Index	.6468	.0072	5.96
8.	Diastolic B.P.	.6479	.0011	0.91
9.	PUSFA	.6491	.0012	1.01
10.	P/SF	.6500	.0009	0.73
11.	SFA	.6510	.0009	0.77
12.	Hard Driving	.6517	.0008	0.64
13.	Systolic B.P.	.6523	.0005	0.44
14.	CHO	.6526	.0003	0.24
15.	Calories	.6530	.0005	0.40
16.	Fiber	.6532	.0002	0.15
17.	Protein	.6533	.0001	0.09
18.	Fat	.6534	.0001	0.05
19.	Cholesterol	.6535	.0001	0.05
20.	Speed & Impatience	.6535	.0000	0.01

After completing the step-wise analysis for the dependent variable SC, the same type of analysis was carried out for the other dependent variable Serum Triglyceride (STG). This was done separately for each age group and then for the total group, taken as whole. From the analysis for 20–29 age group it could be seen that Family History was the first variable to be entered. It accounted for 16 70 per cent of the variance in STG values. The other significant variables in the step-wise solution were obesity index, job involvement, smoking, protein, fat and carbohydrates. They added 6.31, 2.08, 1.81, 1.34, 0.75 and 0.4 per cent respectively. Upto this point the per cent variance accounted was 29.39 per cent only. The total variance accounted by all the variables put together was only 32.93 per cent, leaving the rest unaccounted. Thus, the predictability of STG values in the equation for this age group is rather restricted.

Looking into the step-wise analysis pertaining to 30–39 age group for STG levels, it can be seen that Family History was the major contributor with 17.24 per cent. Smoking was the next variable entered, which contributed 9.83 per cent additionally. This was followed by Job Involvement (3.59), Speed and Impatience (3.02), Age (2.32), dietary cholesterol (2.14), Systolic B.P. (1.76), Calories (1.58), Type A Behaviour (1.75) and Fiber (1.14) in that order. Together these variables contributed to 44.36 per cent of the variance in STG values. The data thus indicate that apart from Family History and Smoking, the other mentioned variables were of minor order. Variables not mentioned above together added only 3.15 per cent (47.51–44.36).

The important variables determining STG values in the 40–55 years age group were, Hard Driving behaviour (9.15 per cent), P/S ratio (4.17 per cent), Family History (4.14 per cent), Fat (3.17 per cent), Sugar (3.1 per cent), Calories (2.57 per cent), Job Involvement (1.46 per cent), SFA (1.26 per cent), Age (1.15 per cent), Type A Behaviour (1.07 per cent), Smoking (1.03 per cent) and Carbohydrates (1.0 per cent). Together, they accounted for 34.15 per cent, while the whole set of variables in the analysis accounted for only 35.09 per cent.

As in the case of SC values, so also for the STG values, a step-wise MRA treating the group as a whole (20–55 years) was done. This data is presented in Table 25. It is seen from the data that age determined 18.95 per cent of the variance, while Family History, Job Involvement, Smoking, P/S ratio and Obesity Index accounted for 9, 4.54, 2.60, 1.07 and 1.05 per cent respectively. Together these variables determined about 36 per cent of the variance, compared to the total variance accounted which was only 39.67 per cent.

3.3.2 Interpretation of Stepwise Contributions

Examining the stepwise MRA for Serum Cholesterol for the three age groups and for the group as a whole, it is clear that the total set of variables accounted for 79.46, 54.41, 63.43 and 65.35 per cent respectively. It is noticed that the three variables Family History, Smoking and Type A Behaviour occupied the first three places (except in the whole group analysis where Age intervened to take the second place). In the 20–29 age group, they accounted for 73.71 per cent of the variance (out of 79.46 per cent totally accounted), while they accounted for 48.12 per cent of the variance (out of 54.41 per cent totally accounted) in the 30–39 age group and 57.31 per cent of the variance (out of 63.42 per cent totally accounted) in the 40–55 age group. Stated in other words, these three variables accounted for about 90 per cent of the variance determined by all the variables in the study put together. From this, it is evident that these three variables viz., Family History, Smoking and Type A Behaviour together become very important predictors of SC levels. Evidently the other variables were of less significance.

At this point, it is relevant to remember that the variable that has the maximum zero-order correlation was entered first. If the later entered variables were correlated with the variables entering earlier, their individual contribution to the variance in the dependent variable gets covered up to the extent of its correlation with the earlier entered variables due to overlap. Only the independent additional contributions (the non-overlapping unique contributions) are indicated as additional variance

Table: 25 Summary Table of the MRA – Stepwise Solution of Serum Triglyceride Values in the 20–55 Age Group

Step No.	Variable Entered	R^2	Increase in R^2	F Value to Enter or Remove
1.	Age	.1895	.1895	69.69
2.	Family History	.2796	.0901	37.15
3.	Job Involvement	.3150	.0454	14.35
4.	Smoking	.3396	.0260	11.92
5.	P/SF	.3503	.0107	4.83
6.	Obesity Index	.3608	.0105	4.82
7.	SFA	.3699	.0090	4.19
8.	Protein	.3751	.0052	2.44
9.	Fat	.3795	.0044	2.04
10.	Hard Driving	.3837	.0042	1.98
11.	Speed & Impatience	.3869	.0032	1.49
12.	Sugar	.3893	.9924	1.15
13.	CHO	.3917	.0024	1.12
14.	Cholesterol	.3934	.0017	0.80
15.	Diastolic B.P.	.3948	.0014	0.64
16.	PUSFA	.3963	.0015	0.70
17.	Fiber	.3965	.0002	0.11
18.	Vitamin C	.3966	.0001	0.10
19.	Calories	.3967	.0000	0.01

contributed by the newly added variable. Thus, in the stepwise analysis for the whole group (20–55 years), Family History and Age were entered as the first two variables and together they took away a large chunk of the variance accounted (i.e. 46.87 per cent out of 65.35 per cent for SC). It is possible that the later entered variables, though they may be important by their own standing, yet, in view of their possible correlation with Family History and Age, their additional contributions get shrunk to the extent of their correlation with Family History and Age (that is to say the common factor variance gets excluded). One way to obviate this difficulty that was available was to blank the first two variables in the MRA, namely Family History and Age and run the analysis. That is to say that the two variables were not included for analysis. By this procedure, variance contributed by the later variables (that was not shown-up to the extent of their overlap with the earlier variables) would come into greater salience than otherwise. This exercise was carried out for both the dependent variables SC and STG, treating the entire sample of 300 subjects as one group.

When the two variables Family History and Age were blanked and the step-wise MRA was re-run for SC, the following factors became more prominent (earlier values are given in brackets). Type A Behaviour 27.74 per cent (4.92 per cent); Smoking 11.12 per cent (10.04 per cent) and Obesity Index 4.32 per cent (0.72 per cent). There was no significant improvement in the other variables.

When this type of analysis was carried out for STG, the following factors became more prominent (earlier values are given in brackets) Job Involvement 12.26 per cent (4.34 per cent); Smoking. 5.24 per cent (2.60); Obesity Index, 4.14 (1.05 per cent); P/S ratio 2.3 per cent (1.07 per cent), Carbohydrate 1.86 per cent (0.24 per cent), Sugar 1.89 per cent (0.24 per cent); and Saturated Fatty acid 1.29 per cent (0.9 per cent).

Evidently, when Family History and Age were taken out of the analysis as independent variables, the variables, Type A Behaviour (or its sub factors), Smoking, and Obesity index seem

to exert more influence both on SC and STG levels. However, in the case of STG, a few dietary variables namely P/S ratio, carbohydrate, sugar, and SFA also became more significant in their contribution to the variance in STG. This latter result illustrates that dietary constituents were also important in influencing the STG levels, though to a lesser degree.

The MRA showed that triglyceride levels were closely associated with positive family history and age. When two independent variables were blanked, the next risk factor that contributed highest to the variance in STG value was Type A Behaviour. This was followed by the obesity index. At this point, it is relevant to note that Albrink (1973) stated that the most important known factors influencing the endogenous plasma triglyceride concentrations were obesity (particularly the adult onset type) dietary carbohydrate and heredity. In the present study too, positive family history contributed significantly to the variance in STG levels of subjects. However, when family history was blanked, obesity index came into prominence adding 4.14 per cent to the value of variance accounted.

The first dietary prescription for hypertriglyceridemia is a reducing diet, which has been shown to successfully lower the TG levels. The observation illustrates the association between obesity and hypertriglyceridemia. Triglycerides increased in healthy subjects who voluntarily raised their body weight by over eating. Excesses of carbohydrates, fat or protein calories are ultimately converted to triglycerides as a matter of economy. Adult onset obesity is more likely to be associated with hypertriglyceridemia. Also, adult onset obesity is associated with large over-filled adipose tissues rather than with too many cells. Perhaps, such oversized cells do not easily take on an even greater triglyceride load, with the result that triglycerides accumulate in serum. Obesity and a high carbohydrate diet contributed to hypertriglyceridemia, while leanness and a low-carbohydrate diet contributed to low triglycerides (Albrink, 1973).

Next, it is seen that the P/S ratio had significantly contributed to the variance in triglyceride levels. Antonis and Bersohn

(1961) showed in all their experiments conducted on white and Banti tribals in Africa that the consumption of diets low in fat calories or high in vegetables oils (i.e. rich in the proportion of highly unsaturated fatty acids), kept serum triglyceride levels low, whereas high fat calorie diets, which were rich in their content of saturated fatty acid, raised the STG levels. The nature of the fat calories in high fat calories diets is therefore of considerable importance in controlling the absolute serum triglyceride level.

Also, there is an interplay between dietary fat and dietary carbohydrates on the effects that compounds have on the concentration and composition of lipids of fasting serum. Sucrose and cream diets elevated the triglyceride levels to a considerable extent (Macdonald, 1967). Hayford *et al* (1973) and Fears *et al* (1981) demonstrated significant increase in fasting plasma triglyceride concentration (51 per cent) during ingestion of sucrose containing diets. Sucrose induced significantly higher plasma triglyceride integrated concentrations than corn syrup diets, whether provided as 45 per cent ($P<0.05$) or as 65 per cent ($P<0.005$) of total energy. Whether this triglyceride response is related to direct substrate effects of fructose on hepatic triglyceride synthesis or related to changes in triglyceride clearance is not resolved and thus the mechanism of sucrose induced increase in circulating triglyceride response is not clearly defined. The significant association between sugar consumption and triglyceride level in the present study confirmed the observations in the reported studies. In this context it is relevant to note that Williams *et al* (1986) mentioned in their study using MRA that the additions of some dietary variables increased the variance accounted by 23.7 per cent in the case of total cholesterol and by 19.9 per cent in the case of TG.

In sum, the MRA of the present study has revealed that of the variables studied, in so far as SC values were concerned, family history, smoking and Type A Behaviour together were the best predictors of SC values in all the three age groups. If family history was out of the picture it was Type A Behaviour and smoking which together predicted 39 per cent with obesity

adding another four per cent. As far as STG was concerned it was again Family History, Smoking and Type A Behaviour that predicted the best except in the 40–55 age group where obesity Index and some dietary variables were added to the list making additional, but only marginal contributions. However, when Family History and Age were taken out, though smoking and Type A Behaviour remained as the more important variables, obesity and a few dietary constituents gained more significance. Further, it was noticed that removal of age and family history from the equation brought down the variance accounted from 65.35 per cent to 48.56 per cent in the case of SC (loss of 16.79 per cent) and from 39.67 per cent to 31.3 per cent (loss of 8.39 per cent) the in case of STG for the whole group.

Compared to other similar investigations, a major thrust of the research reported in the present non-prospective investigation was the examination of the multiple contribution of dietary, non-dietary and coronary prone behavioural components to SC and STG levels in individuals belonging to three age groups 20–29, 30–39 and 40–55. The study assessed the joint determination of these variables as well as their unique contribution to SC, STG levels. In a specific sense, this sort of a study involving dietary constituents and other risk factors including coronary prone behaviour and examining their joint and individual contribution to SC and STG through a step-wise MRA at three age levels was carried out (probably for the first time in India) on a non-hospital population, of course, with some obvious limitations. The analysis was based on a factual study of existing conditions without introduction of any type of manipulation of independent variables in the design. Also, the sample in this study was small (N = 300) and pertained to an university and mostly belonging to middle and upper middle socio economic strata thus restricting its capacity for generalisation to the overall population.

3.3.3 Intercorrelations Among Independent Variables

In most studies, independent variables are correlated among themselves. An examination of these inter-correlations

would give an idea of their inter-relationships. This will be of help in a better understanding of the role of independent variables and their interactions. The inter-correlations among independent variables were assessed.

Several variables were significantly related to increase in Age. They were, Obesity index, Systolic and Diastolic blood pressure, Job Involvement and Hard Driving behaviour. Among dietary variables, calories and proteins were negatively correlated with Age, while PUSFA was positively correlated.

As one grows older, one is likely to become sedantary. However, his food intake does not decrease on par with decrease in activity. This results in a positive energy balance and consequently the weigh/height² goes up. In this study, a correlation of .42 between obesity index and age was observed.

Though a positive correlation was observed between obesity index and Age, calories and protein negatively correlated with age (-.22 and -.34 respectively). This indicates that there was a decrease in protein and calorie intake with age. However the reduction in calories and protein over the years may not be substantial to offset the reduction in activity with Age. This might have contributed to a positive energy balance and there by to obesity.

A positive association was also found between age and systolic and diastolic B.P. (.34 and .88 respectively). It is well-known that several factors (Obesity, Psychological stress, Blood lipids, Sodium etc.) which by themselves may be age associated, also contribute to Hypertension. These, amidst other unknown factors, might have increased B.P. with age.

It is interesting to observe that there is an increase with age of Job Involvement and Hard Driving (both factors of Type A) (r = .26 and .29). The association is low positive but significant. indicating small association. Evidently individuals as they age, appear to become more responsible in their jobs and get involved in their occupation.

The Obesity index was found to correlate with systolic and diastolic B.P., Hard Driving, as well as with Sugar. (r = .29, .47, .26, and .26 respectivel). It is known that Obesity increases blood pressure (Golwalla and Golwalla, 1983; Malhotra and Ganguly, 1976; Nellus *et al*, 1982; and Sambasiva Rao, 1984). Similarly Psychological stress may contribute to over eating (Chandra Patel, 1983) which may inturn lead to obesity. Sugar was shown to increase the body weight (Yudkin, 1972).

Blood pressure correlated with age, Obesity index, and Coronary Prone Behaviour (Hard Driving). As already discussed, blood pressure, both systolic and diastolic increase as age increases and as body weight increases. Certain psychological stresses also put up blood pressure. Chandra Patel (1983); House *et al* (1979) and Kaplan (1972) reported such an association. Family History has been found to relate with Type A Behaviour and smoking significantly (r = .34 and .29 respectively). This shows that quite a few individuals with positive family history of IHD also exhibited Type A Behaviour and were smokers.

Type A Behaviour and its components, speed and impatience, hard driving and job involvement not only correlated among themselves (as would be expected) but also with B.P. and obesity as already mentioned.

Corelations among dietary factors were as follows: calories with carbohydrate, protein, fat and SFA (r = .75, .66, .57 and .25 respectively) significantly; Carbohydrates with protein and fat (r = .52 and .24); Protein with fat (r = .35); Fat with SFA and PUSFA (r = .43, .24 respectively); SFA with PUSFA (.56), P/S (r = .48) and cholesterol (.29) respectively; PUSFA with P/S (.27) and with cholesterol (.23). This indicates that calories are very much related to the three proximate principles i.e., carbohydrate, protein and fat. Naturally the energy is obtained only from the metabolism of these nutrients, most important sources being carbohydrates and fat. Depending on the total calorie content, the distribution would also be in such a way that there is a close correlation between these three

important proximate principles. Fat is constituted by the saturated fatty acid (SFA) and polyunsaturated fatty acids (PUSFA). Therefore it is but natural that there was a significant relationship between the fat and SFA, PUSFA, P/S ratio and cholesterol. PUSFA was correlated with P/S ratio and cholesterol.

The correlations indicate that there was also a significant association between the various fat components and dietary cholesterol. The SFA and dietary cholesterol contribute to an elevation in serum lipids, whereas a high polyunsaturated fatty acid content and thus a high P/S ratio decrease the lipid level. The interaction amongst the dietary variables like fat and cholesterol has been mentioned as the most important of all dietary variables in affecting the cholesterol level (Muller, 1973). The effect of dietary cholesterol on serum cholesterol is controlled by the quality of fat, with SFA enhancing it and PUSFA inhibiting it. A high amount of cholesterol did not elevate the serum cholesterol level, when the diet also had a high amount of linoleic acid on it (Suk Y oh and Monaco, 1985). Thus, the intercorrelation amongst the dietary components is also an important factor that ultimately affects the blood values.

The correlation among the dependent variables SC and STG was also calculated to see whether there is any relationship between the two dependent variables. The value obtained ($r = .33$), which was small but significant, indicates that there was a relationship between the two. This gives a hint that there could be a sort of an abnormality, probably genetic, in the lipid fractions metabolism thus contributing to abnormalities in certain lipoprotein types.

Further, the Very Low Density Lipoprotein (VLDL) catabolism gives way to low Density Lipoproteins (LDL). Therefore, when triglycerides are metabolised, cholesterol is also obtained and that way both may be abnormal in certain individuals.

The foregoing discussion of intercorrelations among independent variables brought out the fact that several variables in the study were intercorrelated. An examination of these intercorrelations helps to understand better the common factor element among the variables in the explanation of variance in the dependent variable variable and gave new gleanings into the interplay of the variables.

Literature is replete with studies that have consistently demonstrated that SC, STG values could be manipulated with benefit by modulation of dietary constituents. If one were to give credence to family history as an important determiner of hyperlipidemia, then dietary control becomes all the more important in these individuals. All the same, the significance of dietary manipulation in the management of hyperlipidemia in individuals who are on the higher side in the risk factor profile can hardly be over stressed. Therefore, dietary manipulation to keep SC and STG values within safe limits especially among those who are susceptible to IHD appears to be a sane and simple prescription.

Granted that dietary intervention can do more good than harm in the control of SC and STG, strategies have to be evolved that could be successful, taking the cheap and easy availability of food material into account in the local Indian context. There is a need to cut the calories commensurate with existing activity at any age level. Simultaneously, application of a dietary regimen is needed. This would be on lines similar to the recommendation of the NIH consensus development conference (U.S.A.) with regard to lipid lowering and IHD prevention (Nut. Rev., 1985).

Dietary habits die hard. Therefore, catch them young and catch them by the forelock are statements that are highly applicable in the development of eating habits of people that would enable the maintenance of lipid levels within limits. Control of quality and quantity of fat intake, increase of fiber, vitamin C and PUSFA in an optimal diet, are considerations of significance. Frequent use of fish foods that are rich in omega-3

PUSFA are bound to be beneficial, in addition to the linoleic acid and its polyunsaturated (omega–6) acids which bring about a negative cholesterol balance. Keeping dietary cholesterol intake within 300 mg/ day is another widely accepted suggestion since this would contain the higher absorption of cholesterol of the "high responders".

It is a common observation that people in the middle and upper socio economic strata frequently consume sweets (eg., Laddu, Mysorepak, Gulab Jamun, Khova etc.) and bakery products (eg, cakes with icings). These contain sucrose and saturated fatty acids in good amounts. The use of non-vegetarian food (mutton and eggs in particular) containing dalda, ghee and animal fat provide rich sources of saturated fat and cholesterol. Though the increased cost of these foods is a restraining factor in their frequent use, yet their consumption by the middle and upper classes would expose them to added risk. The frequent use of fresh fruit (eg, guava, orange, amla etc.), vegetable salads, germinated legumes, curd and other foods containing plenty of fiber and vitamin C. are to be systematically encouraged such that, they could at least partly offset the bad effects of taking rich food.

It would be a good strategy to survey and screen individuals in both urban and rural areas, who have a positive family history of IHD. Such individuals may be monitored and extended dietary counselling by the primary health cenre staff (nutritionist or a dietitian). This is important because positive family history is a major determiner of IHD.

Of equal importance are smokers. Many in the rural areas smoke bidis (leaf cigarettes) and use other forms of tobacco. Smoking is a manipulable risk factor, Urgent steps towards the prevention and discontinuation of smoking needs to be launched on a war footing.

The results of the present study have brought out to a doubtless degree the association of Coronary Prone Behaviour with SC and STG values. The development through reinforcement of the Type B Behaviours coupled with relaxation exercises

and training in crises management on job and off job are realisable goals through sustained effort under guidance. The development of an appropriate philosophy of life that would absorb the stress and strain of modern living should not be a difficult enterprise for the traditional Indian. As such any positive cultural heritage contributing to this end should be encashed. In fact there is a whole gamut of Psychological principles that could be applied towards the acquisition of appropriate non-coronary prone behaviours.

The point has been made time and again that the Indian scenario is fast changing. The culture and life styles of the people, seem to be shifting gradually towards those that would only facilitate the adoption of behaviours and habits of eating that may be broadly termed as coronary risk prone. This observation should be a matter for concern not only among the nation's health keepers but also among its health policy makers. Before it be comes too late in the day and much damage is done, preventive strategies that could work in the Indian setting need to be conceived and executed so that many lives and much expense to the exchequer could be saved. This can come only through widespread public awareness of IHD and its antecedents.

Public knowledge and awareness (through mass education techniques) of the risk factors of IHD in an Indian setting specific to the contemporary living are first steps in prevention. This needs to be followed by development in the public, appropriate attitudes towards prevention and reinforcement of practices that may lead to it. Such efforts should not be isolated but integrated into a package of steps. In this enterprise all the known and proven risk factors of IHD (including hyperlipidemia) need to be simultaneously tackled determining, if necessary, the emphases among them, on the basis of their predictive capabilities (regression weight), coupled with a sensible rationale. In as much as available evidence suggests the laying of IHD foundations in the early decades, preventive policies should cover these groups as well! Such efforts would prevent from perishing, thousands of men in their height of professional life and save much by way of human resource.

In essence, this study has examined and highlighted through MRA, the relative role of several variables, diet and behaviour included, in determination of lipid levels (SC and STG) at three age groups of a sample of middle class, university staff and students. Despite its obvious limitations in view of the selective nature of its sample, yet it has brought out the significant role of several manipulable and non-manipulable factors. It is only a very modest effort, to assess the contributions simultaneously of dietary, behavioural and other classically implicated risk factors of IHD to serum lipids, cholesterol and triglyceride.

Dietary intervention programmes to reduce lipid levels, particularly among the susceptible individuals is of paramount importance in a developing country like India, before it becomes too late.

The foregoing conclusions of this modest effort to analyse the contributions of the several risk factors including Coronary Prone Behaviour aged Dietary constituents to SC and STG levels of a sample of University staff and students can only be extrapolated to the general population with caution. It would be necessary to carryout similar studies on other types of populations and the results confirmed before making generalizations for the general population.

There is a need for experimental studies to examine the relationship between variations in dietary constituents and variations in serum lipids especially serum triglyceride, cholesterol and other lipoprotein fractions among those with positive family history and among the persons with no positive family history. such studies would throw more light on the role of dietary constituents in influencing lipid levels among the coronary prone subjects and among normals.

Studies on effects of therapeutic diets on the blood lipid values in hyperlipidemic individuals need to be carried out and compared with similar Western studies. Such Indian studies can also focus on vegetarian diets and diets that include fish *vis a vis* diets that contain other types of animal foods.

Investigations relating several dietary, behavioural and other risk factors to IHD incidence in India have not been many. Therefore, there is an urgent need to step up research effort in studying these relationships in various parts of India to develop suitable model diets that would reduce the blood lipid levels, if they are higher, and bring down the incidence of IHD. This is particularly important in view of the fact that in the years ahead with greater Industrialization, Urbanization, Modernization and resulting changes in life styles and eating habits, IHD incidence may go up. Efforts to forestall this eventuality are of immediate concern.

After all, the ailing heart has its reason too! It is everybody's business to keep their hearts healthy. A strong heart is the essence of good living. Armed with an arsenal of knowledge on the role of families of factors associated with susceptibility to IHD, one need not lose heart that he would one day *lose* his heart!

REFERENCES

Adlersberg, D. and Shaefer, L.E. (1959) 'The interplay of heredity and environment in the regulation of circulatory lipids and in atherogenesis'. *Am. J. Med., 26,* 1.

Ahrens, E.H. (1985) 'The Diet–heart question in 1985–Has it really been settled?' *Lancet,* 1, 1985.

Ahrens. E.H. Jr., Hirch, J., Oette, K., Febquhar, J.W., and Stein, Y. (1961) 'Carbohydrate–induced and fat induced lipemia'. *Trans Assoc. Am. Physicians,* 74, 134.

Ahrens, R.A. (1974) 'Sucrose, hypertension and heart diseases an historical perspective'. *Am. J. Clin. Nutr.* 27, 403.

Albrink, M.J. (1973) 'Triglyceridemia'. *J. Am. Diet. Ass.* 62, 626.

Anderson, J.W. (1978) 'Effect of carbohydrate restriction and high carbohydrate diets on men with chemical diabetes'. *Am. J. Clin. Nutr.* 30, 402.

Angelico, R. (1975) 'Outlines of dietary prevension of atherosclerosis. In Sirtori. C., Ricci, G. and Gorini, S. (eds)'. *Diet and Atherosclerosis.* New York: Plenum Press.

Antia, F.P. (1973). 'Clinical Dietetics and Nutrition'. Delhi: Oxford Univ. Press.

Apple Bawin–Bowden, D., S.M. Haffner, E. Hartsook, K.H. Luk, J.J. Albers and W.R. Hazzard (1984). 'Down regulation of the low-density lipoprotein receptor by dietary cholesterol'. *Am. J. Clin. Nutr.* [illegible], 360–367.

Arvid Heiberg (1974). 'Heritability of serum lipids and lipoproteins'. In Schettler, G. and Weizel, A. (eds). *Atherosclerosis III.* Proceedings of the 3rd international symposium. Berlin Heidelberg [illegible] Verlag.

Barratt, E.S. (1965). 'Factor analysis of impulsiveness and anxiety.' *Psychological Reports,* 16, 547.

Barrington, H., Abraham, K.A., Hill, P.G., Kanagasabhapathy, A.S. and George, C. (1980). Serum lipids and lipoproteins in control subjects and patients with Ischemic Heart Disease. *J. Assoc. Phys. Ind.* 28. 217.

Barry, L. (1971) Proc. R. Soc. Med. 64, 1005 as given in *Biological Society Symposia,* no. 33, Ed. by Smelle, RMS. p. 28.

Beaglehole, R., Foulkes, M.A., Prior, I.A.M., et al (1980) Cholesterol and mortality in Newzealand Maoris. *Br. Med. J. 1,* 285.

Berkson, D.M. and Stamler, J. (1965) 'Epidemiological findings on cerebrovascular diseases and their implications'. *J. Atheroscler, Res.* 5, 189.

Berry, E.M., Hirsch, J., Most, J., Mc Namara, D.J. and Thornton, J. (1986). 'The relationship of dietary fat to plasma lipid levels as studied by factor analysis of adipose tissue fatty acid composition in a free living population of middle-aged American men'. *Am. J Clin. Nutr, 44,* 220.

Best, C.H. and Taylor, N.B. (1980) *The living body – A text book of Physiology* (4th edn.) London: Chapman and Hall.

Beynen, A.C. and Kafan, M.B. (1985) "Why do polyunsaturated fatty acids lower serum cholesterol?" *Am. J. Clin. Nutr.* 42, 560.

Bhatia, M.L, (1978) Down with high blood pressure, Hypertension in India. *Ind. J. Med. Sci.,* 32, 13.

Blumenthal, J.A., Williams, R., Kong, Y., Schanberg, S.M. and Thompson, L.W. (1978) Type A behaviour and angiographically documented coronary diseases. *Circulation,* 58, 634.

Blumenthal, S., Jesse, M.J., Hennekens, C.H., Klein, B.E., Ferrer, P.E. and Gourley, J.E. (1975) "Risk factors for coronary artery disease on children of affected families". *J. Pediatr.* 98, 1187.

Bordia, A. and Arora, S. (1974) "A comparative study on predisposing factors of Coronary Artery Disease in rural and urban population". *Ind. J. Med. Res.* 62, 565.

Bordia, A., Arora, S.C., Kothari, L.K., Jain, K.C., Rathore, B.S., Rathore, A.S., Dube, C.K. and Bhu, N. (1975). The protective action of essential oils of onion and garlic in cholesterol fed rabbits. *Atherosclerosis,* 22, 103.

B.M.J. (1973) *British Medical Journal* Leading article. 'Obesity and Coronary Heart Disease'. *1,* 566.

B.M.J. (*British Medical Journal*) (1973). Leading Article. "Serum Cholesterol in children". *1,* 690.

Brozek, J., Keps, A. and Blackburn, H. (1966) Personality differences between potential coronary and non-coronary subjects. *Ann. NY. Acad. Sci. 134,* 1057.

Buzzard, M , Mc Roberts, N.R. (1982) "Effect of dietary eggs and ascorbic acid on plasma lipid and lipoprotein cholesterol level in healthy young men". *Am. J. Clin. Nutr. 36,* 94.

Caffrey, B.C. (1968) Reliability and Validity of Personality and behavioural measures in a study of coronary heart disease. *J. Chron. Dis. 21,* 191.

Caffrey et al. (1969) *J. Chron. Dis., 22,* 93.

Carr, J.J. and Drekter, I.J. (1956) 'Simplified rapid technique for extraction and determination of serum cholesterol without saponification'. *Clin. Chem 2,* 353.

Cattell, R.B., Eber, H.W., and Tatsuoka, M. (1970) *Handbook for the sixteen personality factor questionnaire,* Champaign: Institute for Personality and Ability Testing.

C.F.T.R.I. Publication (1976). *Balanced diets and nutritive value of common recipes.* Mysore; C.F.T.R.I., 84–92.

Chandra Patel (1983) A new dimension in the prevention of coronary heart disease. In Dembroski, T.M., Weiss, S.M., Shields, J., Haynes, S., & Feinleib, M. (eds) *Coronary Prone Behaviour,* New York: Springer.

Cohen, J. (1968) Multiple regression as a general data analytic system *Psychological Bulletin, 70,* 426.

Connor, W.E., [illegible] (1961) "The serum lipids in men receiving high cholesterol and cholesterol-free diets". *J. Clin. Invest.* 40, 894.

Dahlstrom, W.G. and Welsh, G.S. (1960) *An MMPI Handbook: A guide to use in clinical practice and research.* Minneapolis; University of Minnesota Press.

Davidson, S.S., Passmore, R., Brock, J.F. and Truswell, A.S. (1975) *Human Nutrition and Dietetics,* (6th edn.) Livingstone; The English Language Book Society.

Dawber, J.R. and Nickerson, J.R. (1982) "Eggs, serum cholesterol and coronary heart disease". *Am. J. Clin. Nutr.* 36, 617.

DeBakey, M.E., Gotto, A.M., Scott, L.W. and Foreyt, J.P. (1986) "Diet, nutrition and heart disease". *J. Am. Diet. Assn.* 86, 729.

Dembroski, T.M., Mc Dougall, J.M., Shields, J.L., Petitto, J. and Lushene, R.L. (1978) Comparants of Type A coronary – Prone behaviour pattern and cardiovascular responses to Psychomotor Challenge. *J. Behav. Med.* 1. 159.

Devadas, R.P. (1979) "*Diets for prevention of atherosclerosis*" Regional Workshop on 'Planning diet for health'-Manual, W.C.C., Madras, E-13-19.

Devadas, R.P., Anuradha, V. and Sheela, R. (1980) "Dietary pattern and serum cholesterol levels of selected Tamilian and Gujarati Women". *Ind. J. Nutr. Dietet.* 17, 159.

Devadas, R.P. and Eswaran, P.P. (1979) "*Diets for prevention of atherosclerosis*". Regional workshop on planning diet for health – Manual, W.C.C., Madras, E. 13.

Dhar, S.N., Shaw, S.N.A., Shafi, M., Yousuf, M., Nafae, A. and Khan, G.G. (1978) Incidence and pattern of heart disease in a hospital population in Kashmir. *J. Asso. Phys. Ind.* 26, 567.

Dunbar, H.F. (1943) *Psychosomatic Diagnosis.* New York; Hoeber.

Elmadjian, F., Hope, J.M. and Lamson, E.T. (1958) Excretion of epinephrine and norepinephrine under stress. *Recent Prog. Har. Res.* 14, 513.

Epstein, F.H. (1964) "Hereditary aspects of coronary heart disease". *Am. Heart J.,* 67, 445.

Eysenck, H.J. and Eysenck, S.B.G. (1968) *Eysenck Personality Inventory.* San Diego: Educational and Industrial Testing Service.

Fears, R., Glenny, R.P., Tredger, J.A. & Lindsay, R. (1981). 'Sucrose induced hypertriglyceridemia: Relation to HDL–C and to physical fitness'. *Nut. Rep. Internati,* 24, 909.

Feinleib, M., Brand. R.J., Remington, R. and Zyzanski, S.J. (1978) Association of the coronary prone behaviour pattern and coronary heart disease. In Dembroski, T.M., Weiss, S.M., Shields, T.L., Haynes, S.G. and Feinleib, M. (eds.), *Coronary Prone Behaviour.* New York: Springer.

Fidanza, F. (1972) "*Dietary studies and Epidemiology of heart disease*". Proc. 1st Asian Congress of Nutrition, NIN, ICMR., Nat. Soc. India.

Fidanza, F. (1972) "Epidemiological evidence for the fat theory". *Proc. Nutr. Soc.* 31, 317–321.

Fillios, L.C. and Mann, G.V. (1954) *Metabol.* 3, 16.

Fletcher, M.N. (1968) 'A Colorimetric method for estimating Serum Triglycerides'. *Clin. Chem. Acta.* 22, 393.

Forde, O.H., Knutsen, S.F., Arnesen, E. and Thelle, D.S. (1985) "The Troms heart study; Coffee consumption and serum lipid concentrations in men with hypercholesterolemia; a randomised intervention study. *Br. Med. J.* 290, 893.

Foster, L.B. and Dunn, R.T. (1973), Stable reagents for determination of Serum Triglycerides by a Colorimetric Hantzsch Condensation method, *Clin. Chem.* 12, 338.

Fredrickson, D.S., Levy, R.I. and Lees, R.S. (1967) Fat transport in lipoproteins an integrated approach to mechanisms and disorders. *N. Engl. J. Med.* 276. 32, 94, 148, 215 and 273.

Friedman, M., Byers, S.O. and Brown, A.E. (1967) Plasma lipid responses of rats and rabbits to an auditory stimulus. *Am. J. Physiol. 212,* 1174.

Friedman, M., Byers, S.O., Diamant, J. & Rosenman, R.H. (1975), Plasma catecholamine response of coronary prone subjects (Type A) to a specific challenge. *Metabol., 4,* 205.

Friedman, M., ST. George, S. and Byers, S.D. (1960) Excretion of catecholamines, 17–Ketosteroids, 17–hydroxycorticoids, and 5–hydroxyindole in men exhibiting a particular behaviour pattern (A) associated with high incidence of clinical coronary artery disease. *J. Cli. Invest., 39,* 758.

Friedman, M. and Rosenman, R.H. (1959) Associates of specific overt behaviour pattern with blood and cardiovascular findings. *J. Am. Med. Ass., 169.* 1286.

Friedman, M., Rosenman, R.H. and Carroll, V. (1958) changes in the serum cholesterol and blood clotting time in men subjected to cyclic variation of occupational stress. *Circulation, 17,* 852.

Tripp, R.R., Hodgson, J.L., Kwiterovich, P.O., Werner, J.C., Schuler, H.G. and Whifman, V. (1985). "Aerobic capacity, Obesity and Atherosclerotic risk factors in male adolescents". *Pediatrics, 75,* 813.

Gandhi, B.M. (1982) Lipoprotein composition of normal health subjects in northern India. *Ind. J. Med. Res. 75,* 393.

Garden, H. and Eugene, I. (1983). *Harrison's Principles of Internal Medicine.* London: Mc Graw Inc., Book Company.

Gertler, M.M., White, P.D., Cady, L.D. and Whiter, H.H. (1964) "Coronary heart diseases". *Am. J. Med. Sci. 248,* 35.

Ginter, L. (1974) *Vitamin C–Recent aspects of its Physiological and Technological importance. Vitamin C in lipid and Atherosclerosis.* London: Applied Science Publishers Ltd.

Glass et al., (1977) *Am. Sci. 65,* 177.

Glueck, C.J., Gordon, D.J., Neison, J.J., Davis, C.E. and Tyroler (1986) "Dietary and other correlates of changes in total and low density lipoprotein cholesterol in hyper cholesterolemic men". The lipid research clinics coronary primary prevention trial". *Am. J. Clin, Nutr, 44,* 489.

Gafman, J.W., and Young, W. (1963) "The filtration concept of atherosclerosis and serum lipids in the diagnosis of atherosclerosis. In Sandler, M. and Bourne, G.H. (Eds.) *Atherosclerosis and its Origin.* New York: Academic Press, New York.

Goldberger, L. and Breznitz, S. (1982) *Handbook of Stress: Theoretical and Clinical Aspects.* New York: The free Press.

Gopalan, C., Ramasastri, B.V. and Balasubramanian. S.C. (1985) "*Nutritive value of Indian Foods*" New Delhi: Indian Council of Medical Research Publication.

Goto (1974) Dietary management of hyperlipoproteinemia. In Schettler, G. and Weizel, A. *Atherosclerosis III,* Berlin Heidelberg: Springer – Verlag.

Gotto, A.M. and Scott, L. (1973) "Dietary aspects of Hyper-triglyceridemia" *J. Am. Diet. Ass., 62,* 617.

Gough, H.G. (1956) *California Psychological Inventory.* Palo Alto. Consulting Psychologists Press.

Gough, H.G. and Heilbrun, A.B. (1980) *The adjective checklist* Palo Alto: Consulting Psychologists Press.

Grande, F. (1967) Dietary carbohydrates and serum cholesterol. *Am. J Clin. Nutr. 20,* 176.

Grande, F., Anderson, J.T., Chlouve Vakis, C., Proja, M. and Keys, A. (1965). "Effect of dietary cholesterol on man's serum lipids". *J. Nutr. 87,* 52.

Gupta, S.P., Gupta, M.S. and Moga, R.C. (1980) "Smoking as a coronary risk factor–comparative evolution of cigarette paper and bidi leaf". *Ind. J. Med. Sci. 34,* 163.

Gupta, S.S., Siwach, S.B. and Moda, V.K. (1979) "Age and Sex related trends in blood pressure in the general population of Haryana". *Ind. J. Med. Res.*, *69*, 834.

Gupta, P.R., P. Khetrapal and C.L. Ghai (1987) "Effect of Garlic on serum cholesterol and electrocardiogram of rabbit consuming normal diet". *Ind, J. Nutr. Dietet. 41*, 6.

Hammond, E.C., Garfinkel, L. and Seidman, H. (1972) "Longevity of Parents and grand parents in relation to coronary heart disease and associated variables". *Circulation, 43*, 41.

Harold, P.M. and J.E. Kinsella (1996) "Fish oil consumption and Decreased risk of cardio vascular disease: a comparison of findings from animal and human feeding trials". *Ame. J. Clin. Nutr. 43*, 566–598.

Harper, H.A., Rodwell, V.W. and Mayes, P.A. (1977) "*Review of Physiological chemistry: Metabolism of Lipids* (17th ed.). London: Lange Medical Publications.

Hartman, G. (1974) Dietary management of hyperlipoproteinemias-calories, In Schettler, G. and Weizel. A. (eds.) *Atherosclerosis III*. Berlin Heidelberg: Springer Verlag.

Hatco, F.T. (1974) "Interaction between nutrition and heredity in CHD". *Am. J. Clin. Nutr.* 27, 80.

Hayford, J T., Daney, M.M., Wiebe, M.C.D., Roberts, S. and Thompson, R.G. (1979) "Triglyceride integrated concentrations: effect of variation of source and amount of dietary carbohydrate". *Am. J. Clin. Nut. 32*, 1670.

Hegsted, D.M. (1986) "Serum cholesterol response to dietary cholesterol: a re-evaluation" – Special Article. *Am. J. Clin Nutr. 44*, 299.

Hegsted, D.M., McGandy, R.B., Myers, M.L., and Stane, F.J. (1965). "Quantitative effects of dietary fat on serum cholesterol in man". *Am. J. Clin. Nutr. 17*, 231.

Hennekens, C.H., Jesse, M.J. and Klein, B.E. (1976) "Cholesterol among children of men with myocardial infarction". *Pediatrics*, 58, 211.

Fletcher, M.N. (1968), A Colorimetric method for estimating serum triglycerides *Clin. Chem. Acta, 22*, 343.

Hillman, L.C, Porters, S.G., Fisher, C.A. and Pomare, E.W. (1985). "The effects of the fiber components pectin, cellulose and lignin on serum cholesterol levels". *Am. J. Clin. Nutr. 42*, 207.

House, J.S., Mc Michael, A.S., Wells, J.A., Kaplan, B.H., and Landerman, L.R. (1979) Occupational stress and health among factory workers. *J. Heal. Soc. Beh., 20*, 139.

ICMR (1984). *Recommended Dietary Intake for Indians* New Delhi: ICMR Publication.

ICMR Bulletin (1986). *Diet and Atherosclerosis*, vol. 16(9) 101–105. New Delhi: Division of Publication and Information, ICMR.

Indrayan, A., Srivastava, R.N. and Bagchi. S.C. (1972a) "Influence of some correlates of blood pressure on its distribution in an adult urban population of Allahabad". *Ind. J. Med. Res. 60*, 651.

Indrayan, A., Srivastava, R.N. and Bagchi, S.C. (1972b) "Age regression of blood pressure in an urban population of 15–19 years. *Ind. J. Med. Res. 60*, 966.

Insel, P.M. and Moos, R.H. (1974) *Work Environment Scale*. Palo Alto: Consulting Psychologists Press.

Jamuna, D. & Sujatha Ramamurti (1984) Coronary-Prone Behaviour as a function of age. *Psychological Studies, 29*, 165.

Janabai Giri, J.K., Sakthi Devi and Meera Rani, S. (1984) Effect of ginger on serum cholesterol levels. *Ind, J, Nutr. Dietet. 21*, 433.

Jenkins, C.D. (1966), Components of the coronary prone behaviour pattern: their relation to silent myocardial infarction and blood lipids. *J. Chron. Dis. 19*, 599.

Jenkins, C.D. (1971), Psychological and Social precursors of coronary disease, *New Engl. J. Med., 294*, 987.

Jenkins, C.D. (1976) Recent evidence supporting psychologic and social risk factors for coronary disease. *New Engl. J. Med. 294,* 987.

Jenkins, C.D. (1982) Psychological risk factors for coronary Heart Disease. *Acta. Med. Scad.,* 660, 123.

Jenkins, C.D., Hames. C.G., Zyzanski, S.J., Rosenman, R.H. and Friedman, M. (1969). Psychological traits and serum lipids. Part I: Findings from the California Psychological Inventory. *Psychosom Med., 31,* 115.

Jenkins, C.D., Rosenman, R.H., and Friedman, M. (1961) Replicability of rating the coronary-prone behaviour pattern. *Br. J. Pre. Soc. Med., 22,* 16.

Jenkins, C.D., Rosenman, R.H., and Zyzanski, S.J. (1968) Cigarette Smoking: Its relationship to coronary heart disease and related risk factors in the Western Collaborative group study. *Circulation, 38,* 1140.

Jenkins, C.D., Rosenman, R.H., and Zyzanski, S J. (1974) Prediction of clinical coronary heart disease by a test for the coronary-prone behavioural pattern. *New Engl. J. Med., 290,* 1270.

Jenkins, C.D., Rosenman, R H., and Zyzanski, S.J. (1979) *Jenkins activity Survey.* New York: Psychological Corporation.

Jenkins C.D., Zyzanski, S.J. and Rosenman, R.H. (1971) Progress toward validation of a computer scored test for the type A coronary-prone behaviour pattern. *Psychosom, Med., 33,* 193.

Jenkins, C.D., Zyzanski. S.J. and Rosenman, R.H. (1976) Risk of new myocardial infarction in middle aged men with coronary heart disease. *Circulation, 3,* 61.

Johnson, C.C., Epstein, F.H. and Kjelsberg, M.O. (1965) "Distributions and familial studies of blood pressure and serum cholesterol levels in a total community-Tecumseh, Michigan". *J. Chron. Dis., 18,* 147.

Judd, J.T., Marshall. M.W. and Canary, J.J (1983). *Changes in blood pressure and blood lipids of adult men consuming modified fat diets. Human nutrition research.* Simpson Gerber and Philadelphia Cary Gerber & Company.

Juneja. A. and Sharma, I. (1981) A comparative study of effect of dietary habits on serum lipids in middle aged normal and heart patient males. *Ind. J. Nutr. Dietet. 18,* 300.

Kannel, W.B., Kagan, A., Revotskie, N. and Stokes, J. (1961) "Factors of risk in the development of coronary heart disease-six year follow up experiences". *Ann. Intern. Med., 55,* 33.

Kannel, W.B. (1974) "*The role of cholesterol in coronary atherogenesis*, Atherosclerosis. *Medical Clinics of North America.*

Kannel- W.B., Castelli, W P., Gordon, T., McNamara, P.M. (1971). 'Serum Cholesterol, lipoproteins and the risk of CHD'. *Ann. Intern. Med. 74,* 1.

Kannel, W.E. (1977a) "Preventive Cardiology" what should the clinician be doing about it? *Post-graduate Medicine, 61,* 74.

Kannel, W.B. (1977)[b] "Prevention of heart disease in the young coronary candidate". *Primary Care, 4,* 229.

Kannel, W B. and Gordon, T. (1973) *The Framingham Study. An epidemiological investigation of cardiovascular disease* section. 29. DHEW Publication (NIH) 74–478.

Kannel, W.B. and Gordon, T. (1980) Cardiovascular risk factors in the Aged: *The Framingham Study.* Proc. of the 2nd conference on the Epidemiology of Aging. Haynes, S.G. and Fenleib. M. (eds.) NIH. Publication (USDH) No. 80-969, 65–89.

Kannel, W.B., LeBauer, E J, T.R. Dawber, M.E., and Mc Namara, P.M. (1964) Relation of body weight to development of coronary heart disease". *Circulation, 35,* 734.

Kaplan, R.D. (1972) Organisational stress and individual strain: A socio-psychological study of risk factors in coronary heart disease among administrators, Engineers and Scientists. *Dis. Abs. Intl.*, *32*, (11B), 6706B.

Kaplan, B.H., Cassel, J.C., Tryder, H.A., Cornoni, J.O., Kleinbaum. D.G. & Hames, C.G. (1971) Occupational mobility and coronary heart disease. *Arch. Intern. Med.*, *128*, 936–942.

Kare Berg (1989) 'Role of genetic factors on atherosclerotic disease? *Ame. Jr. of Clin. Nutr. 49*, 1027–29.

Karvonen, M.J. (1972) "Modification of the diet in primary prevention trials. "*Proc. Nutr. Soc.*, *31*, 355.

Kay, R.M. and Straberg, S.M. (1978) "Origin, Chemistry, Physiological effects and Clinical importance of dietary fibre". *J. Clin. Invest. Med.*, *1*, 9.

Kay, R.M., Z.I. Sabry, and A.C. Sima (1980) Multivariate analysis of diet and serum lipids, *Amer. Jr. of Clin. Nutr.*, *33*: 2566–2572.

Kay, R.M. and Truswell, A.S. (1977). "Effect of citrus pectin on blood lipids and fecal steroid excretion in man". *Am. J. Clin. Nutr. 30*, 171.

Keith, R.L., Lown, B. and Stare, P.J. (1965) Coronary heart disease and behaviour patterns: an examination of method. *Psychosomatic Medicine*, *27*, 424.

Kenigsberg, D., Zyzanski, S.J., Jenkins, C.D., Wardwel, W.I. and Licciardello. T.A. (1974). The coronary prone behaviour pattern in hospitalized patients without coronary heart disease. *Psycho. Med. 36*, 344.

Kerlinger, F.N. and Pedhazur, J.E. (1973) *Multiple Regression in Behavioural Research.* New York; Holt, Rinehart and Winston, Inc.

Keys, A. (1965) "Serum Cholesterol response to changes in the diet". *Metab. Clin. Exp. 14*, 747.

Keys, A. (1970) *Coronary Heart disease in seven countries*, American heart association monograph. No. 29; Pub, by Am. Hear. Asso., Inc.

Keys, A. (1984) Serum cholesterol response to dietary cholesterol. *Am. J. Clin. Nutr.*, *40*, 351.

Keys, A. (1986) "Serum cholesterol response to dietary cholesterol" Letters to Editor. *Am. J. Clin. Nutr. 44*, 309.

Keys, A., J.T. Anderson, and Grande, F., "Serum Cholesterol response to changes in the diet II". The effect of cholesterol in the diet. *Metabol. 14*, 759.

Keys, A., Aravanis, C., Blackburn, H., Vanbuchen, F.S.P., Buzina, R., Djordjenic, B.S., Fidanza, F., Karvonen M.J., Menotti, A., Puddu, V. and Taylor, H.L. (1972) Probability of middle aged men developing coronary heart disease in 5 years. *Circulation, 45*, 815.

Keys, A., Taylor, H.L., Blackburn, H., Brozek, J. and Anderson, J.T. (1971) Mortality and Coronary Heart disease among men studied for 23 years. *Ann. Internal Med.*, *128*, 201.

Kittel, F., Dornitzer, M., Zyzanski, S.J., Jenkins, C.D., Rustin, R.M. and Degre, C. (1978) Two methods of assessing the Type A Coronary – prone behaviour pattern in Belgium. *J. Chron. Dis.*, *31*, 147.

Kozarevic, D., Pire, B., Ravic, Z., Dawber, T.R., Gordon, T. and Zykel, W.J. (1976). The Yugoslavia Cardiovascular disease study: Part II: Factors in the incidence of coronary heart disease. *Am. J. Epidemiol. 104*, 133.

Kretch, [illegible] (1979) Some aspects of bile acid and urobilinogen excretion and fecal elimination in men given [illegible] and egg formulas with and without added bran. *Am. J. Clin. Nutr. 32*, 1492.

Krishnaswami, S., Richard, J., Sathyamurthy, I., Babu Uthaman, C. and Sukumar, I.P. (1980) Multivariate analysis of risk factors in coronary artery disease – Predictive value in [illegible] *Ind. J. Med. Res.*, *79*, [illegible]

Kritchevsky, D. (1982) Fiber and Lipids. In Vahouny, G.V. and Kritchevsky, D. (Eds.): *Dietary fiber in Health and Disease*. New York: Plenum Press.

Kromhout, D. (1983) "Body weight, diet, and serum cholesterol in 871 middle aged men during 10 years of follow-up. (The Zutphen study). *Am. J. Clin. Nutr. 38,* 591.

Krumdick, C. and Butterworth, C.E. (1974) "Ascorbate cholesterol lecithin interaction – factors of potential importance in the pathogenesis of atherosclerosis". *Am. J. Clin. Nutr. 20,* 808.

Kumar, M., Chakravarthi, R.N., Singh, A. and Wahi, P.L. (1976) "Serum lipid profiles in patients with myocardial infarction in the Chandigarh area (Northern India). *Atherosclerosis, 24,* 355.

Lacombe, C,R., G.R. Comraze and M.M. Nibbelink. (1986), 'Effects of a low energy diet associated with egg supplementation on plasma cholesterol and lipoprotein levels in normal subjects, results of a cross-over study'. *Bri. J. Nutr. 56,* 561–575.

Lamberg, S.L. and Rothstein, R. (1978) *Hematology and Urinalysis* Functional medical laboratory technology. A comprehensive series of Manuals. AVI Publishing Company, INC.

Lanza, E and Butrun, R.R. (1986) "A critical review of food fiber analysis and data". *J. Am. Diet, Ass. 86,* 732.

Lapidus, L., Anderson, H., Bengtsson, C and Bosaeus, I. (1986). "Dietary habits in relation to incidence of cardiovascular disease and death in women: A 12 year follow up of participants in the population study of women in Gothenburg, Sweden"; *Am. J. Cli. Nutr. 44,* 444.

Less, R.S (1974). Workshop-Dietary management of hyper lipidemias-Calories in Schettler, G. and Weizel, A. *Atherosclerosis,* III. Berlin Heidelberg: Springer Verlag.

Leibowitz, J.O. (1970) *The History of Coronary Heart Disease.* Berkeley: University of California Press.

Levine, R S., Hennekens, C.H and Rosmer, B. (1978) 'Aggregation of cholesterol among young families of men with myocardial infarction". *Am. J. Epidemiol. 108:* 227.

Levy, R.J. (1973) "Triglycerides as a risk factor in Coronary Artery disease". *J. Am Med. Ass, 224,* 1770

Lewis, B., Katau, M. and Merkx, M. (1981) "Towards an improved lipid-lowering diet: additive effects of changes in nutrient intake". *Lancet,* 2, 1310.

Liebman, M. and Bazzare, T.L. (1983) "Plasma lipids of vegetarian and non-vegetarian males: effects of egg consumption". *Am. J. Clin. Nutr. 38,* 612.

Little, J.A., Shanoff, H.M., Caima, A, and Yano, R. (1966) "Coffee and serum lipids in coronary heart disease". *Lancet, 1,* 732.

Liu, G C., Coulston, A M. and Reaven, G M. (1983) "Effect of high carbohydrate, low fat diets on plasma glucose, insullin and lipid responses in hypertriglyceridimic human". *Metabol.* 32, 750;

Lovallo, W R. and Pishkin, V (1980) A Psychophysiological comparison of type A and B men exposed to failure and uncontrollable noise. *Psychophysiology, 17,* 29.

Macdonald, I. (1967) "Interrelationship between the influences of dietary carbohydrates and fats on fasting serum lipids". *Am. J. Clin. Nutr., 20,* 345.

Macdonald, I (1970) "*Relationship between dietary carbohydrates and lipid metabolism*", Nutrition Dieta. Symposium Publ. *15,* 129.

Macdonald, I. (1975) Diet and human Atherosclerosis – Carbohydrates: In Sirtori, C., Rissi, G. and Gorini, S. (Eds), *Diet and Atherosclerosis* New York: Plenum Press.

Mann, G.V and White, H.S. (1953) The influence of stress on plasma cholesterol levels. *Metabol.* 2, 47.

Mancini, M. (1974) Workshop. Dietary management of hyperlipoproteinemia in: Atherosclerosis III. Proceedings of International Symposium. Eds. Schettler, G. and Weizer, A. Springer Verlag Berlin, Heidelberg. New York, 763.

Marmot, M.G. (1984) "Geography of blood pressure and hypertension". Br. Med. Bulletin, 40, 380.

Marmot, M G. and Syme, S.L. (1976) Acculturation and coronary heart disease in Japanese-Americans. *Am. J. Epidemio, 104,* 225

Martin, D.W., Mayes, P.A. and Rodwell, V.W. (1981) *Harper's Review of Biochemistry* 17th ed. of Lipids cholesterol metabolism. Lange Medical Publishers Marugen Asia Ltd.

Mathur, K.S. (1973) "Prevention of coronary heart disease". *Ind. J. Med. Sci. 27,* 226.

Mathur, K.S. (1983) 'Dietary factors and Serum Lipids' Ind. Heart, Jour. 35, 250.

Mattson, F.H. and Grundy, S.M. (1985) "Comparison of effects of dietary saturated, monounsaturated and polyunsaturated fatty acids on Plasma lipids and lipoproteins in man". *J. Lip. Res. 26,* 194.

Mc Donough, J.R., Hames, C.C., Greenberg, B.G., Griffin, L.H. and Edwards, A.J. (1962) Observations on serum cholesterol levels in the twin population of Evans county, Georgia". *Circulation, 25,* 962.

McGill, H.C. Jr. (1974) The Lesion, In Schettler, G. and Weizel, A. (Eds). *Atherosclerosis* III. Berlin Heidelberg Springer Verlag.

McGill, H.C Jr. (1979) "The relationship of dietary cholesterol to serum cholesterol concentration and to atherosclerosis in man *Am. J. Clin. Nutr.* 32, 12.

McGill, H.C., Gees, J.C. and Strong, J.P. (1903) *Atherosclerosis and its origin;* New York: Academic Press.

Mc Intosh, G.H., Richmond, W., Himsworth, R.L. (1981) "Vitamin C deficiency and hypercholesterolemia in Marmoset monkeys". *Nutrition Reports International, 23,* 237.

McMichael, J. (1979) Fats and arterial disease. *American Heart Journal, 98,* 409.

Mcnamara, D.J., R. Koeb, T.S. Parker, H. Batwin, P. Samuel, C.D. Brown and E.H. Ahrens (1987) 'Heterogeneity of Cholesterol homeostasis in man - Response to changes in Dietary fat quality and cholesterol quantity'. J. Clin. Invest. 79, 1729-1739.

McNemar, Q. (1960) At random: Sense and Non-sense. *American Psychologist, 25,* 295.

Medalie, J.H., Kahn, M.A., Neufeld, H.N. et al (1973) Myocardial infarction over a five year period: I Prevalence, incidence and mortality experience. *J. Chron. Dis., 26,* 63.

Medeiros, D.M. (1982) 'Caffeinated beverage consumption and blood pressure in Mississipi young adults" *Nut. Rep. Int. 26,* 563.

Miettinen, J.A. (1971) Cholesterol production in obesity. *Circ, 44,* 842.

Moore, M.C., Cruxaman, M.A.. Chilling, P.E. and Strong, J.P. (1977) "Dietary - Atherosclerosis; study on diseased persons". *J. Am. Diet. Ass. 70,* 602.

Moore D.J., J. White, P.R. Halt and D.V. Parke (1985) 'Beneficial short term effects of unprocessed wheat bran on lipid and glucose metabolism in man'. *Hum. Nut. Cli. Nut. 39c,* 63-67.

Moses, C. (1963) "*Atherosclerosis, Mechanisms as a guide to prevention*". Philadelphia: Lea and Febiger.

Muller, J.F. (1973) "A dietary approach to coronary artery disease". *J. Am. Diet. Ass., 62,* 613.

Murchison, L.E. (1985) "Clinical Algorithms" - Hyperlipidemia. *Br. Med. J. 290,* 535.

National Institutes of Health (1979) The MRFIT behaviour study. Part I: Study design, procedures, and reproducibility of behaviour pattern judgements *J. Chron. Dis., 32,* 293.

NIN Bulletin (1984) "*Some common Indian Recipes and their Nutritive value*". Hyderabad: NIN.

Nestel, P.J., Hevenstein, N., Whyte, H.M., Scott, T.J. & Cook, L.J. (1973) Lowering of plasma cholesterol and enhanced sterol excretion with the consumption of polyunsaturated ruminant fats. *New Engl. J. of Medicine, 288,* 379.

Nestel, P.J., Verghese, A. and Lovell, R.R.H. (1967) Catecholamine secretion and sympathetic nervous responses to emotion in men with and without angina pectoris. *American Heart Journal, 73,* 227.

Nestel, P.J. (1986) 'Fish oil attenurates, The cholesterol induced rise in Lipoprotein cholesterol'. *Ame. Jr. Clin. Nut. 43,* 752–757.

Nestel, P.J. (1978) "Poly unsaturated Fatty acids" *Am. Jr, Clin. Nut. 45,* 1161–1167.

NIH Consensus Development Conference Statement (1985)– Lowering Blood Cholesterol to prevent Heart Disease: A special report. *Nut. Rev. 43,* 283–285.

Nirmala, K. Murthy, Parvatham, R. and Jeyanth, G.P. (1986) Biochemical changes in elderly women belonging to a low socioeconomic group. *Ind. J. Nut. Diet, 23,* 31.

Nissinen, A. and Stanley (1989) 'Unbalanced Diets as a cause of chronic diseases' *Ame. Jour. Clin. Nut, 49,* 993–8.

Norgan, N.A. and Ferrolitzi, A. (1982) "Weight, height indices as estimates of fatness in men". *Hum. Nut. Clin. Nut. 36,* 363.

Nutrition Today. (1970) 'More about Sucrose and Atherosclerosis". *Nut. Today, 5,* 15.

Nut. Rev. (1985) *Body weight and serum cholesterol. 43,* 43.

Nut. Rev. (1985) *Male pattern obesity as a risk predictor for a coronary heart disease, stroke and death. 43,* 45.

Opie, L.H. (1973) "Lipid metabolism of the heart and arteries in relation to IHD". *Lancet,* 1, 192.

Osborne, R.H., and Adlersberg, D. (1958) "Serum lipids in twins" *Science, 127,* 1294.

Osborne, R.H., Adlersberg, D., DeGeorge. F.V. and Wang, C. (1959) "Serum lipids, heredity and environment". *Am. J. Med. 26,* 54.

Pasi, W.P., Rothfeld, B., Isom, K,E. and Varady, A. (1973) Cholesterol synthesis and metabolism as a function of unpredictable shock stimulation. *Physiol & Behav. 11,* 107.

Pelletien, O. and Keith, M.O. (1974) "Availability of synthetic and natural ascorbic acid". *J. Am. Diet. Ass, 64,* 271.

Pietinen, P., Vartainen, E., H.J. Korhonen, L. Kartovaara, U. Vusilalo, J. Tuomilehto and P. Puska (1989) 'Nutrition as a component in community control of cardiovascular disease (The North Karelia Project)'. *Ame. Jour. Cln. Nutr. 49,* 1017–24.

Pinto, I.J., Thomas, P., Colaco, F. and Datey, K.K. (1970) Current development in India. Section X. Environmental and host factors CHD, including risk factors – An epidemiological view. In Jones, R.J. (ed). *Atherosclerosis.* Proceedings of 2nd International Symposium.

Posner, B.M., DeRusso, P.A., Norquist, S.L. and Erick, M.A. (1986) "Preventive nutrition in coronary heart disease: Risk assessment and formulating dietary goals". *J. Am. Diet. Ass. 86,* 1395.

Prior, I. (1978) "Down with high blood pressure – How natural is hypertension. The Pacific riddle. *Ind. J. Med. Sci., 32,* 8.

Pyorala, K. (1989) "Dietary cholesterol in relation to plasma cholesterol and CHD". *Ame. Journ. Clin. Nutr. 45,* 1176–84.

Raab, W. (1953) *Hormonal and neurogenic cardiovascular* disorders. Baltimore: Williams and Wilkins.

Raab, W. (1966) Prevention of Ischemic Heart Disease – Principles and practice springfield: Charles C. Thomas.

Ramamurti, P.V., Jamuna, D. and Sujatha Ramamurti (1984) A study of coronary – prone behaviour among a sample of executives and non executives. *Ind. J. Clin. Psychol. 11,* 75.

Rahe, R.H., Rubin, R.T. and Arthur, R.J. (1974) The three investigators study: Serum Uric acid, cholesterol, and Cortisol variability during stresses of everday life. *Psychosom. Med. 36,* 258.

Rahe, R.H., Rubin, R.T., Gunderson, E.K.E, and Arthur, R.J. (1971) Psychologic correlates & serum cholesterol in man: A longitudinal study. *Psychosom, Med. 33,* 399.

Reiser, R., Probstfied, J., Silvers, A., Scott, L.W., Shorney, M.L., Wood, R.D., Obrien, B.C., Gotto, Jr. A.M. and Insull, W. (1985) Plasma lipid and lipoprotein response of humans to beef fat, coconut oil and safflower oil. *Am. J. Clin, Nutr. 42,* 190.

Rifkind, B.M. (1986) Issues and Opinions in Nutrition - Diet, plasma cholesterol and coronary heart *Disease J. Nutr. 116,* 1578.

Rifkind, B.M., Goor, R.S. and Levy, R.J. (1979) *Current status of the role of dietary treatment in the prevention and management of coronary heart disease.* Vol. 63. The Medical Clinics of North America. Philadelphia: W.B. Saunders Company.

Rifkind, B,M. and Segal, P. (1983) Lipid Research Clinics programme: reference values for hyperlipidemia and hypolipidemia. *J. Am. Med. Ass., 250,* 1869.

Rissanen, A.M. and Nikkila, E.A. (1977) Coronary artery disease and its risk factors in families of young men with angina pectoris and in controls. *Br. Heart J. 39,* 875.

Robert Scheig (1974) Diseases of lipid metabolism In Bondz Rosenberg, Duncan (Ed.) *Diseases of Metabolism.*

Robertson, D., Frolich, J.C., Keith, C., Watson, J.T., Hollifield, J.W., Shand, D.G. and Oates, J.A. (1978) Effects of Caffeine on plasma renin activity, Catecholamines and blood pressure. *New Engl. J. Med. 298,* 181.

Robertson, J.I. (1978) Doing something about high blood pressure *Ind J. Med. Sci,, 32,* 35.

Robertson, W;B. and Strong, J.P. (1968) Atherosclerosis in persons with hypertension & diabetes Mellitus, McGill, H C. Jr. (Ed): *Geographic Pathology of* atherosclerosis. Baltimore, Williams & Williams Company.

Robinson, C H. (1967) *Proudfit-Robinson's normal & Therapeutic nutrition.* Calcutta: Oxford and India Book House.

Robinson, C.H. (1977) *Normal and Therapeutic nutrition* New York : Macmiltan & Co.

Roine, P, (1972) *Dietary prevention of Coronary heart disease.* Proc. 1st Asian Congress of Nutrition, Nut. Soc. India, Hyderabad.

Rose, G.A. and Blackburn, H. (1968) *World Health Organisation* Monograph Series - No. 56, Cardiovascular Survey Methods, WHO, Geneva.

Rosenman, R.H. (1978) The interview method of assessment of the coronary prone behaviour pattern, In Dembroski, T.M, Weiss, S.M., Shields, J.L., Haynes, S.G. and Feinleila, M. (eds.), *Coronary prone behaviour* New York: Springer.

Rosenman, R.H. (1978a) Role of Type A Behaviour pattern in the pathogenesis of Ischemic heart disease and modification for prevention. *Advances in Cardiology, 25,* 1.

Rosenman, R.H. (1978b) The role of the type A behaviour pattern in Ischemic heart disease: Modification of its effects by beta - blocking agents. *Br. J. Clin. Prac., 32,* 58.

Rosenman, R.H. (1983) Current status of risk factors and Type A Behaviour pattern in the pathogenesis of Ischemie Heart Disease. In Dembroski, T.M., St. Peterburg, F., Schmidt, T.H., Blumchen, G.C. Leichingen (eds). *Biobehavioural bases of Coronary Heart Diease,* New York: Karger Biobehavioural Medicine (Series-2, 262.

Rosenman, R.H., Brand, R.J., Sholtz, R.I. and Friedman, M. (1976) Multivariate prediction of coronary heart disease during 8.5 year follow-up in the Western collaborative group study. *Am. J. Cardiol. 37,* 903.

Rosenman, R.H., Brand, R.J., Sholtz, R.I., Friedman, M., Straus, R. and Wurm. M. (1975) coronary heart disease in the Western Collaborative Group Study: Final follow-up of 8 5 years. *J. Am. Med. Ass. 233,* 872,

Rosenman, R.H. and Chesney, M.A. (1980) the relationship of Type A behaviour pattern to coronary heart disease. *Activitas Nervose Superior, 22,* 1.

Rosenman, R.H. and Friedman, M. (1961) Association of specific behaviour pattern in women with blood, and cardiovascular findings. *J. Am. Med. Ass . 24,* 1173.

Rosenman, R.H. and Friedman, M. (1963) Behaviour patterns, blood lipids, and coronary heart disease, *J. Am. Med. Ass. 184,* 934.

Rosenman, R.H. and Friedman, M. (1974) Neurogenic factors in the Pathogenesis of coronary heart disease. *Medical Clinics of North America, 58,* 269.

Rosenman, R.H., Friedman, M., Strans, R., Wurm, M., Kositchek, R., Hahn, W. and Werthessen, N.T. (1964) A predictive study of coronary heart disease: The Western Collaborative Group Study: *J. Am. Med. Ass, 189,* 15.

Rosenman, R.H., Rahe, R.H., Borhani, N.O. and Feinleib, M. (1976) Heritability of personality and behaviour. *Acta Genetical Medical et Gemellogiae, 25,* 221.

Sainani, G.S. and Mehta, P.J. (1984) Are we Really Behind the West. *J. Ass. Phys, India, 32,* 237.

Salans, L.B., Cushman, S.W. and Weisman, R.E. (1978) Studies of human adipose tissue; adipose cell size and number in non-obese patients *J. Clin. Invest., 52,* 929.

Samuel, P. and Shalchi, O.B. (1964) Effect of vitamin C on serum cholesterol in patients with hyper-cholesterolemia and atherosclerosis. *Nut. Abs. and Rev., 34,* 1104.

Sapru, R.P. (1984) A lowest estimate of the prevalence of cardiovascular disease in India. *J.A.P.I., 32,* 251.

Schacky, C.T., S. Fischer and P.C. Weber (1985) Long Term effects of Dietary marine W-3 fatty acids upon plasma and cellular lipids, platelet function and eicosanoid formation in humans. *J. Clin. Invest, 76,* 1626–1631.

Schettler, F.G. and Boyd, G.S. (1969) *Atherosclerosis* Amsterdam: Elsevier Publishing Company.

Schilling. F.J., Christakus, G.J., Bennet, N.J. and Coyle, J.F. (1964) Studies of serum cholesterol in 4244 men and women an epidemiological and pathogenetic interpretation. *Am. J, Pub. Health, 54,* 461.

Seltzer, C.C., and Mayer, J. (1264) Body build and obesity – who are the obese: *J. Am. Med. Ass. 189,* 677,

Senthilanathan, C. (1979) *Nutrition in cardiovascular disease and in Atherosclerosis* Regional Workshop Manual on Planning Diet for Health. W.C.C., Madras. P.E.-3.

Seymour, Dayton (1975) Nutrition and Atherosclerosis *Progress in Food and nutrition Science, 1,* 191.

Shaefer, L.E., Adlersberg, D. and Steinberg, A. (1958) Heredity, environment and serum cholesterol: *Circulation, 17,* 537.

Shafer, C.F. (1970) Ascorbic acid and atherosclerosis *Am. J. Clin. Nutr. 23,* 27.

Shanmuga Sundaram, K.R., Viswanathan, A., Dhandapani, K. Srinivasan, N., Rosappan, Gilbert, R., Alladi, S., Kanchela, S. and Vasanthi, N. (1986). Effect of high fat diet on cholesterol distribution in plasma lipoproteins, esterifying activity in leucocytes and erythrocyte membrane components studied; importance of body Wt. *Am. J. Clin. Nut. 44,* 805.

Shaper, A.G. (1972) Diet in the epidemiology of coronary heart disease. *Proc. Nut. Soc., 31,* 297.

Shekelle, R.B., Schoenberger, J.A., Stamler, J. (1976) Correlates of the JAS Type A behaviour Score. *J. Chron. Dis., 29,* 381.

Simopoulos, A.P. (1965) The health implications of over weight and obesity. *Nut. Rev., 43,* 33.

Simpson, M.T., Olowine, D.A., Jenkins, C.D., Ramsey, F.H., Zyzanski, S.J., Thomas, G. and James, C.G. (1974) Exercise induced catecholamines and platelet aggregration in the coronary prone behaviour pattern. *Psychosom. Med. 34*, 476.

Singh, S P., Sisodia, A.K., Rizvi, S.I.B., Jain and P.N. Jain (1980) A study of the effect of dietary habits on total serum cholesterol level in young healthy adults. *Ind. J. Nutr. Dietet, 17*, 216.

Sinha, A.K., Shyam Sundar, K,, Vyas, S.K. (1985) Effects of smoking on blood pressure, body weight, electrocardiogram and serum cholesterol in young hypertensives. *Cloniwan, 49*, 41.

Sirtori, C., Ricci, G. and Gorini, S. (1975) *Diet and Atherosclerosis. Advances in Experimental Medicine and Biology.* Vol *60*, Plenum Press, New York and London.

Skyring, A., Modan, B., Croeeth, A. and Hammerstrom, C. (1963) Some epidemiological and familial aspects of coronary heart disease – Report of a pilot study. *J. Chro. Dis. 16*. 1267.

Slater, C.H., Sonith. D.P, Nichaman, M. and Slattery, M. (1985) Ischemic heart disearse – Foot prints through the data. *Am. J, Clin. Nutr. 42*, 329.

Sogani, R.K. and K Katoch (1981) Correlation of serum cholesterol levels and incidence of myocardial infarction with dietary onion and garlic eating habits. *J. Asso, Phy. Ind. 29*, 443.

Sokoloff, B.. Hon, M., Saelhof, C.C., Wrzolek, T. and Imai, T. (1966) Aging–Atherosclerosis and Ascorbic acid metabolism. *J. Am, Genet. Soc. 14*, 1239.

Sommariva, D., Scott, L. and Fasoli, A. (1978) Low fat versus low carbohydrate diet in the treatment of type IV. hyperlipoproteinemia. *Atherosclerosis, 29*, 43.

Soni, G.L., George, M. and Singh, R. (1982) Role of common Indian pulses as hypo cholesterolemic agents *Ind. J. Nutr. Dietet, 19*, 184.

Sood, A.K., Umesh Kapil and Gupta, M.C. (1985) Epidemiology of Obesity in an urban community. *Ind, J. Nut. Dietet. 22*, 42.

Spielberger, C.D., Gorsueh, R.L. and Lushene, R.E. (1970) *State-trait anxiety inventory.* Palo Alto: Consulting Psychologists Press.

Spittle, C.R. (1972) Atherosclerosis and Vitamin C. *Lancet,* 1335.

Srikantaiah, S.G., Jagannathan, S.N. and Gopalan, C. (1961) Serum cholesterol and blood pressure levels in some South Indian Population groups. *Ind. J. Med. Res 49*, 99.

Srimathi, V., Seshagiri, P.B., Raju, R. and Ramakrishnan, S. (1981) Effect of coffee and tea on cholesterol and triacyl glycerol of blood serum of humans and rats. *Ind. J. Nut. Dietet, 18*, 360,

Steptoe, A. (1981) *Psychological factors in cardiovascular disorders:* New York, Academic Press.

Stamler, J., Berkson, D.M., Lindberg, H.A., Hall, Y., Miller, H., Mojonnier, L., Levenson, M., Cohen, D.B., Young, Q.D. (1966). Coronary Risk Factors – Their importance and therapy in the prevention of CHD. *The Medical Clinics of North America, 50*, 229.

Stone, M C. (1972). Role of diet in the management of hyperlipoproteinemias. *Proc. Nut., Soc. 31*, 511.

Story, J.A. and Thomas, J.N. (1982). Modification of Bile Acid Spectrum by dietary fiber. In Vahounif, G.V. and Kritchevsky, D. *Dietary Fiber in Health and Disease.* New York: Plenum Press.

Strong, J.A., Eggen, D.A., Oatman, M.C., Richards, M.L. and Tracy, R.E. (1978) Pathology and epidemiology of Atherosclerosis. *J. Am. Diet. Ass. 62*, 262.

Suk, Yi Oh and Miller, L.T. (1983). Effects of dietary egg on variability of plasma cholesterol levels and lipoprotein cholesterol. *Am. J. Clin. Nutr, 52*, 421.

Swaminathan, M. (1974). *Essentials of Food and Nutrition.* Vol. *I* Fundamental Aspects, Madras: Ganesh & Co.

Swanson, J.E., J.M. Black and J.E. Kinsella (1988). Dietary mehaden oil: effects on the rate and magnitude of modification of phospholipid fatty acid composition of mouse heart and Brain. *Brit J. Nut. 59,* 535–545.

Syme, S.L., Hyman, M.M. and Enterline, P.E. (1964). Some social and cultural factors associated with the occurence of coronary heart disease. *J. Chron. Dis. 17,* 277.

Syme, S.L., Hyman, M.M. and Enterline, P.E. (1965) Cultural mobitity and the occurence of coronary heart disease. *J. Heal. Soc. Behav. 6,* 178.

Taskar, S.P., Iyyer, I.R., Nesurkar, S.V., Sawant, P.N., Bhansale, D.R. and Kinare, S.G. (1983). Lipid profile in patients with myocardial infarction in the city of Bombay, *Ind. Heart. Jour. 35,* 169.

Thelle, D.S., Arnesen, E. and Forde, O.H. (1983). The Troms Heart Study. Does coffee raise serum cholesterol ?. *New Engl. J. Med. 308,* 1454.

Thelle, D.S., Armsen, E. and Forde, O.H. (1984). Coffee and serum cholesterol, *Br. Med. J. 388,* 1960.

Thensen, L., L.B. Henriksen and P. Engby (1986). One year experience with low fat low cholestesol diet in patients with coronary heart disease. *Am. J. Clin. Nutr. 44,* 212–219.

Thiele, F.V. (1980). *Clinical Nutrition* London: C.V. Mosby Company, 2nd ed.

Thurstone, L.L. (1949). *Thurstone temperament schedule* Chicago; Science Research Associates.

Tillotson, J.L., Winston, M.C. and Hall, Y. (1984). Critical behaviours in the dietary management of hypertension. *J. Am. Diet. Ass. 84,* 290.

Topping, D.L., Hood, R.L. and Okenfull, D.G. (1977) Dietary saponins and plasma cholesterol. *Proc. Nutr. Soc. 38,* 78A.

Tzagournis, M. (1978). Triglycerides in Clinical Nutrition Perspectives in Nutrition. *Am. J. Cli. Nutr. 31,* 1437.

Uma, B. and Chakrabarti, C.K. (1973). Effect of supplementation of some essential aminoacids on tissue levels of cholesterol and phospholipid of albino rats fed different pulse proteins. *Ind. J. Nut. Dietet. 10,* 68.

Usha, C., Geetha, G. and Shanti, V. (1983). Effect of raw and roasted bengalgram on some physiological parameters in albino rats. *Ind. J. Nut. Dietet. 20,* 40.

Vahouny, G.V. (1982). Dietary fibers and Intestinal Absorption of Lipids. In Vahouny, G.V. and Kritchevsky, D. (Eds). *Dietary fiber in Health Disease.* New York: Plenum Press.

Vajpayee, A., Chadda, V.S , Jain, N.C., Mishra, S.N., Suleman, A A. and Singhvi, D R. (1981) Serum lipids and smoking habit in first degree relatives of patients of IHD. *Ind. Heart. J. 33,* 274.

Van Dusch, T. (1968). *Lehbuch der Herzkrankheiten.* Liepzig: Engleman.

Varley, H. (1975). *Practical Clinical Biochemistry.* Arnold Heinemann Publishers, New Delhi.

Vergroesen, A.J. (1972). Dietary fat and cardiovascular disease: Possible modes of action of linoleic acid. *Proc. Nutr. Soc. 31,* 323.

Vessby, B., Lithell, H. and Boberg, J. (1982) Reduction of Low density and high density lipoprotein cholesterol by fat modified diets. A survey of recent findings. *Hum. Nutr. Clin. Nutr. 36,* 203.

Vigue, J.L., D. Lairon, P. Borel, H. Portugal, A.M. Pauli, T.C. Hanton and H. Lafond (1987). Effects of Pectin, wheat bran and cellulose on serum lipids and lipoprotein, in rats fed on a low or high fat diets. *Brit. J. Nut. 58,* 405–413.

Vijayalakshmi, P. and Lakshmi, V. (1985). Biochemical profile of smokers and nonsmokers. *Ind. J. Nutr. Dietet. 22,* 62.

Vijayammal, P.L., S. Lelemma, K. Premakumari and P.A. Kurup. (1982). Effect of composition of diet on some aspects of cholesterol and triglyceride metabolism in rats. *Ind J. Med. Res. 75*, 868–875.

Voudoukis, I,J. (1971). Exaggerated cold pressor response in patients with atherosclerotic vascular disease, *Angiograph, 22*, 57.

Vranic, M. (1975) Turnover of free fatty acids and triglyceride – A overview. *Fed. Proc. 34*, 2233.

Wald. N. Howard. S., Smith. P.G. and Jeldren. K. (1973). Association between atherosclerotic disease and carboxy haemoglobin levels to tobacco smokers. *Br. Med. J. 1* (31st March 1. 76.

Waldron, I.. Nowotarski, M,. Preimer, M.. Henry, J.P., Post, N, and Witten. C. (1682). Cross-cultural variation in blood pressure: A quantitative analysis of the relationships of blood pressure to cultural characteristics, salt consumption and body weight. *Soc, Sci. Med. 16*, 419.

Waldron, I., Zyzanski, S., Shekelle, R,B,, Jengins. C.D. and Tannebaum, S. (1977). The coronary – prone behaviour pattern in employed men and women. *Journal of J. Human Stress, 3*, 2.

Wells. V,M, and Bronte, S.B, (1963). Egg yolk and serum cholesterol levels – Importance of dietary cholesterol intake. *Br. Med. J. 1*, 577.

Werner. G.I. and Sareen, D.K. (1978). Serum Cholesterol levels in the population of Punjab in North West India. *Am. J. Clin. Nutr. 31*, 1479.

W.H.O. Bulletin (1970). Beaumont, J.L., Carlson, L.A., Cooper. G.R., Feijfar, Z., Fredrickson, D.S., Strasser, T. Classification of hyperlipidemias and hyperlipoproteinemias. *Bull WHO* (1970) *43*, 891.

Whitney, E.N. and Cataldo, C.B. (1983) *Understanding Normal and Clinical Nutrition.* St. Paul: West Publishing Company.

William, S.R. (1969). *Nutrition and Diet Therapy* St. Louis; C.V. Mosby Company.

Williams, H.B., Karen, S., Feldt, B.N. and Feiberl, J.H. (1983) Community surveillance of stroke in persons under 70 years old – contribution of uncontrolled hypertension. *Am, J. Pub. Heal. 73*, 260.

Williams, P.T., Krause, R.M., Joyce, S.K., Dreon, D.M., Vranizan, K.M. and Wood, P.D. (1986). Relationship of dietary fat, protein, cholesterol and fiber intake to atherogenic lipoproteins in men. *Am. J Clin. Nut. 44*, 788.

Wilson, C. (1969). Hypertension In Schettler, F.G. & Boyd, G.S. (eds). *Atherosclerosis*. Amsterdam; Elsevier Publishing Co.

Whol, M.G. and Goodhart, R.S. (1968). *Modern Nutrition in Health and Disease.* Philadelphia: Lea and Febigec.

World Health Organisation, (1978). Report of the WHO expert committee on Hypertension. *Tech. Rep.* Ser. No, 625. Geneva.

Yudkin, J. (1964). Dietary fat and dietary sugar in relation to Ischemic heart disease and diabetes. *Lancet, 4*.

Zyzanski, S.J. and Jenkins, C.D. (1970). Basic dimensions within the coronary – prone behaviour pattern. *J. Chron. Dis., 22*, 781.

Zyzanski, S.J., Jenkins, C.D., Ryan, T.J., Elessas, A. and Everist, M. (1976). Psychological correlates of coronary angiographic findings, *Archives of Internal Medicine, 136*, 1234.

Zyzanski, et al (1979). *Soc. Sci. Med., 13A*, 405.

APPENDIX — I

Diet Questionnaire

Name :

1. Are you a vegetarian or Non-vegetarian ?

2. How often do you eat the following foods ?

S.No.	Item	FREQUENCY Daily	2 or 3 times a week	Weekly once	Once 2 weeks	Once month
1.	Eggs					
2.	Mutton/Beef/Pork or organ meat (specify)					
3.	Chicken					
4.	Fish/Prawns					
5.	Greens-leafy vegetables					
6.	Other vegetables Brinjal beans etc.					
7.	Potatoes – Yam etc,					
8.	Citrus fruits (Oranges, sweet limes etc.)					
9.	Plantains					
10.	Butter/Ghee					
11.	Curd					

3. a) Do you like cream (in milk) and butter ? If yes, how often do you eat these.

   b) Do you like roasted foods better than boiled foods, eg., curries.

   c) How much dalda/ghee do you consume a day?

4. Do you eat onion/Garlic : If yes, how much.

Kindly record the amount of food consumed by you during a 3 day period in terms of the cup provided to you.

	First day	Second day	Third day
	Cups or No.	Cups or No.	Cups or No.
Breakfast			
Give name and quantity in either number or terms of cup.			
Tea / Coffee / Other beverage (give name and quantity).			
Total cups / day.			
Lunch			
Rice			
Curry (non-vegetarian or name of vegetable)			
Sambar			
Rassam			
Curd/Butter milk Tea–snacks if any.			
Dinner			
Rice			
Curry (give the name of the vegetable)			
*OR Non-vegetarian food			
*Sambar (Mention vegetable)			
Chutney			
Rasam			
Buttermilk/curd			
Fruit			

If you take any other foods or drinks other than above, mention them and quantity taken (Eg. Tea time snacks)

*The Non-vegetarians may please mention the preparation and quantity.

What type of oil and how much was used per day to prepare the above menus? Please mention the correct quantity (in terms of cup).

Please mention the quantity of dalda or ghee, if used to prepare any of the recipe mentioned above.

APPENDIX — II

NUMBER — 1

Smoking Questionnaire

1. (a) Do you smoke cigarettes now?
   Yes, regularly/No. occasionally

   (b) Do you inhale into lungs? Yes/No

   (c) What kind of cigarettes do you smoke?
   Manufactured with filters.
   Manufactured without filters.
   Hand-rolled or Beedies.
   Cigars.

   (d) How many manufactured cigarettes/Beedies/Cigars do you usually smoke per day?

   (e) What is the maximum number of cigarettes that you have smoked per day for as long as a year?
   (record total number of manufactured and hand-rolled cigarettes, counting 1 oz of tobacco as 25 cigarettes and 1 g as 1 cigarette).

   (f) How many cigarettes did you smoke per day a year ago?

   (g) How old were you when you began to smoke cigarettes?

2. (a) If not did you ever smoke cigarettes in the past.
   Yes, regularly/No/Occasionally (less than one)

   (b) What is the maximum number of cigarettes you ever smoked per day for as long as a year?
   (record total number of manufactured and hand-rolled cigarettes, counting 1 oz of tobacco as 25 cigarettes and 1 g as 1 cigarette).

   (c) Did you inhale? Yes/No

   (d) How old were you when you began to smoke cigarettes?

   (e) When did you stop smoking cigarettes?
   ([illegible] year)

   (f) Why did you stop?

NUMBER — 2

Initial Examination

Is your Mother alive? Yes/No

If yes, does she have heart disease?

If no, (a) What did she die of?

(b) How old was she when she died?

Is your Father alive? Yes/No

If yes, Does he have heart disease? Yes/No

If no, (a) What did he die of?

(b) How old was he when he died?

Your Father's occupation:

How many brothers and sisters have you who are alive now?

Number of Brothers alive: No. of Sisters alive:

Have any of them ever had heart trouble? Yes/No/Not applicable

If yes, please give details as follows:

Brother or Sisters	Age when trouble started	Nature of illness
1.		
2.		

Did you have any brothers or sisters who died? Yes/No

If yes, please give the following information for each one:

Brother or Sister	Age	Cause of death
1.		
2.		
3.		

How many children have you had (incl[illegible]y who died):

If you have had children, how many have died?

NUMBER — 3

Chest Pain on Effort

1. Have you ever had any pain or discomfort in your chest? Yes/No

1a. If 'No', have you ever had any pressure or heaviness in your chest? Yes/No

If 'No', proceed to section C.
If 'Yes' ask next question (if during the remainder of section A an answer is recorded in a box marked*, proceed to section B).

2. Do you get it when you walk uphill or hurry? Yes/No
Never hurries or walks uphill–

3. Do you get it when you walk at an ordinary pace on the level? Yes/No

4. What do you do if you get it while you are walking?
Stop or slow down/Carry on

5. If you stand still, what happens to it? Relieved/Not relieved*

6. How soon? 10 minutes or less/More than 10 minutes*

7. Will you show me where it was? Sternum (Upper or middle)
(Record all areas mentioned) Sternum (lower)
Left arm
Other

8. Do you feel it anywhere else? Where? Yes/No
(If 'Yes', record additional information above)

9. Did you see a doctor because of this pain (or discomfort)? Yes/No

If 'Yes', what did he say it was?

Section — B : Possible Infarction

10. Have you ever had a severe pain across the front of your chest lasting for half an hour or more Yes/No
    (If 'Yes', ask question 11)
11. Did you see a doctor because of this pain ? Yes/No
    If 'Yes', what did he say it was ?
    How many of these attacks have you had ?
    1st attack : Date : Duration of pain :
    2nd attack : Date : Duration of pain :

Section — C : Intermittent Claudication

If an answer is recorded in a box marked*, no further questions need be asked.

12. Do you get pain in either leg on walking ? Yes/No*
13. Does this pain ever begin when you are standing still or sitting ? *Yes/No
14. In what part of your leg do you feel it ?
    Pain includes calf/calves/pain does not include calf/calves
15. Do you get if you walk uphill or hurry ? Yes/No*
    Never hurries or walks uphill
16. Do you get it if you walk at an ordinary pace on the level ? Yes/No
17. Does the pain ever disappear while you are walking ? *Yes/No
18. What do you do if you get it when you are walking ?
    Stop or slow down/Carry on
19. What happens to it if you stand still ?
    Relieved/Not relieved
20. How soon ?
    10 minutes or less/More than 10 minutes.

NUMBER — 4

Dyspnoea Questionnaire

If disabled from walking by any condition other than heart or lung disease, mark X here and leave remaining questions unanswered :

1. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill ? YES/NO
   If 'NO', stop here. If 'YES', proceed to next question.
2. Do you get short of breath walking with other people of your own age on level ground ? YES/NO
   If 'NO' stop here. If 'YES', proceed to next question.
3. Do you have to stop for breath when walking at your own pace on level ground ? YES/NO
   If 'NO' stop here. If 'YES', proceed to next question.
4. Are you short of breath on bathing washing or dressing ? YES/NO

NUMBER — 5

Bio-Data

1. a) Name :
   b) Date of birth and age in Years :
   c) Education :
   d) Postal address :
   e) Occupation :
   f) Father's and Mother's occupation ;

g) Monthly income (tick the appropriate) :

a) Rs. 500 – 1000 ;
b) Rs. 1000 – 1500 ;
c) Rs. 1500 – 2000 ;
d) Rs. 2000 – 2500.

2. a) Did your father/mother suffer from heart disease. Yes/No – Fatherr/Mother

b) If yes give details : Nature of disease
Age of onset
Serum cholesterol level
Triglyceride level
Blood pressure
Diabetes (sugar)

3. a) Give the relationship of other near relation who had heart disease.
1. Uncles
2. Aunts
3. Brothers/Sisters
4. Grandfather/Grandmother

b) Nature of heart disease :

4. a) Do you play regularly : YES/NO
If yes state game played :

b) Do you do exercise or walking regularly : Yes/No
If Yes state duration or distance :

5. Do you consume alcohol : Yes/No
If Yes how often : Rarely/Regularly/Occasionally

6. Do you have diabetes : Yes/No

7. Your height – Weight : a) Height :
b) Weight :
c) W/H :

8. Blood pressure reading :

9. Blood lipids : Cholesterol
Triglyceride
HDL